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TO: Celine Qian **Location: REM-2A64/2C70** **Art Unit: 1636** **Wednesday, January 19, 2005** **Case Serial Number: 10/009579**	**From: Edward Hart** **Location: Biotech-Chem Library** **REM-1A55** **Phone: 571-272-2512** **edward.hart@uspto.gov**

Search Notes

Examiner Qian,

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Please feel free to contact me if you have any questions.

Edward Hart

WEST Search History

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	DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L15	L8 and L14	4
☐	L14	L13 same (promoter or regulat$ or UTR or enhancer)	16
☐	L13	human pancarcinoma associated epithelial glycoprotein-2 or EGP-2 or 17-1A or Ep-CAM	519
☐	L12	human pancarcinoma associated epithelial glycoprotein-2 or EGP-2 17-1A or Ep-CAM	95
☐	L11	L8 near5 (promoter or regulat$ or UTR or enhancer)	67
☐	L10	L8 same (promoter or regulat$ or UTR or enhancer)	145
☐	L9	L8 and (promoter or regulat$ or UTR or enhancer)	1006
☐	L8	carcinoma near3 (specific or select$ or restrict$)	1553
	DB=PGPB,USPT,USOC,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L7	L5 same lung carcinoma	0
☐	L6	L5 and lung carcinoma	18
☐	L5	L4 same (promoter or regulat$ region or regulat$ element or 5 prime UTR)	87
☐	L4	carcinoma near3 (select$ or specific$ or prefer$)	1683
☐	L3	L1 near3 (promoter or regulat$ region or regulat$ element or 5 prime UTR)	1
☐	L2	L1 and (promoter or regulat$ region or regulat$ element or 5 prime UTR)	307
☐	L1	EGP-2 or Ep-CAM or 17-1A or GA733-2	573

END OF SEARCH HISTORY

ATTN: Ed Hart

142604

Access DB# _____

SEARCH REQUEST FORM

CRFE

Scientific and Technical Information Center

Requester's Full Name: Celine Qian (STIC) Examiner #: 78770 Date: 1/13/04

Art Unit: 1636 Phone Number ~~30~~ 2-0777 Serial Number: 10/009,579

Mail Box and Bldg/Room Location: 2A64 Results Format Preferred (circle): ~~PAPER~~ DISK E-MAIL

If more than one search is submitted, please prioritize searches in order of need.

****

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of invention: Non-Squamous Epithelium-Specific Transcription

Inventors (please provide full names): Lou, De Leij et al.

Earliest Priority Filing Date: 3/1/2000

For Sequence Searches Only Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.

Please search SEQ ID NO:5 From 3115 bp - 3560 bp.
(-778 to -442 figure 1)

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PTO-1590 (8-01)

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         alerts (SDIs) affected
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         alerts (SDIs) affected
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awareness
         alerts (SDIs) affected
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         alerts (SDIs) affected
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         February 2005
 NEWS 17 JAN 11 CA/CAPLUS - Expanded patent coverage to include Russia
         (Federal Institute of Industrial Property)

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         MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
         AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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=> s EGF 2 or epithelial glycoprotein 2 or Ep CAM or 17 1A or GA733 2
L1        1526 EGF 2 OR EPITHELIAL GLYCOPROTEIN 2 OR EP CAM OR 17 1A
OR GA733 2

=> s l1 and (promoter or regula? element or regulat? region or 5 UTR)
L2          52 L1 AND (PROMOTER OR REGULA? ELEMENT OR REGULAT?
REGION OR 5
          UTR)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3          37 DUP REM L2 (15 DUPLICATES REMOVED)

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YOU HAVE REQUESTED DATA FROM 37 ANSWERS - CONTINUE? Y/(N):y

L3  ANSWER 1 OF 37  CAPLUS  COPYRIGHT 2005 ACS on STN
AN  2004:1020014  CAPLUS
DN  142:5477
TI  Recombinant virus expressing an intact anti-tumor antibody containing
    human immunoglobulin constant regions and the therapeutic use thereof
IN  Qian, Qijun; Yang, Qin
PA  Sino-Gene Biotechnology Ltd., Peop. Rep. China
SO  PCT Int. Appl., 50 pp.
    CODEN: PIXXD2
DT  Patent
LA  Chinese
FAN.CNT 1
    PATENT NO.         KIND  DATE       APPLICATION NO.      DATE
    ---------------    ----  --------   -------------------  --------
PI  WO 2004101777      A1    20041125   WO 2004-CN430        20040429
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
           CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
           GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
           LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
           NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
           TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
        RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
           AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
           EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
           SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
           SN, TD, TG
PRAI CN 2003-116733     A   20030430
AB  The present invention provides a recombinant virus comprising a chimeric
    gene which encodes an intact anti-tumor antibody contg. human Ig const.
    regions and the therapeutic use thereof. By inserting into the genome of
    a recombinant virus a nucleotides acid sequence which simultaneously
    comprises. The cDNA sequences of both light chain and heavy chain gene of
    an intact anti-tumor antibody with human Ig const. regions are inserted
    into the genome of a recombinant virus, and the intact anti-tumor antibody
    can be expressed in tumor cells with high efficiency, thereby inhibit the
    growth and metastasis of tumors. In particular embodiments, the cDNA
    expressing human anti-EGFR antibody, or humanized antibody specific to
    human Her2, and chimeric human-mouse anti-CD20 antibody are prepd. and
    inserted into a replication deficient adenovirus. The anti-tumor activity
    of chimeric human-mouse anti-CD20 antibody is tested in breast cancer cell
    line BT-474 and a nude mouse implemented with breast cancer cell line
    SK-OV-3.
RE.CNT  4   THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
RECORD
          ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3  ANSWER 2 OF 37  CAPLUS  COPYRIGHT 2005 ACS on STN
AN  2004:927015  CAPLUS
DN  141:394059
TI  Human EpCAM or TAg-25, fragments, chimeric derivatives, antibodies and
    conjugates for cancer diagnosis and therapy
IN  Punnonen, Juha; Apt, Doris; Neighbors, Margaret; Leong, Steven R.
PA  Maxygen, Inc., USA
SO  PCT Int. Appl., 273 pp.
    CODEN: PIXXD2
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.         KIND  DATE       APPLICATION NO.      DATE
    ---------------    ----  --------   -------------------  --------
PI  WO 2004093808      A2    20041104   WO 2004-US12280      20040419
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
           CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
           GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
           LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
           NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
           TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
        RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
           BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
           ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI,
           SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,
           TD, TG
PRAI US 2003-464780P    P   20030422
AB  The invention provides novel polypeptides, including novel tumor-assocd.
    antigens, and related nucleic acids, vectors, cells, fusion nucleic acids
    or polypeptides, ligands and antibodies. The invention also provides
    compns. comprising such polypeptides, nucleic acids, vectors, cells, and
    antibodies, and methods of producing and using the same.

L3  ANSWER 3 OF 37  CAPLUS  COPYRIGHT 2005 ACS on STN
AN  2004:902213  CAPLUS
DN  141:378849
TI  Immunogenic recombinant antibodies for use as vaccines against infection,
    autoimmune disease and cancer in primate such as human
IN  Loibner, Hans; Himmler, Gottfried; Waxenecker, Guenter; Schuster, Manfred;
    Putz, Thomas
PA  Igeneon Krebs-Immuntherapie Forschungs- und Entwicklungs-A.-G., Austria
SO  PCT Int. Appl., 59 pp.
    CODEN: PIXXD2
DT  Patent
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LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004091655	A2	20041028	WO 2004-EP4059	20040416

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI AT 2003-599 A 20030417

AB The invention refers to an immunogenic recombinant antibody designed for immunization of primates comprising at least a part of a murine IgG2a subtype amino acid sequence and a mammalian glycosylation. The antibody is a chimeric, humanized, monoclonal, anti-idiotypic, or bi-isotopic antibody or fragment. The antigen is an epitope or mimotope of tumor-assocd. antigen, epithelial cell adhesion mol., Lewis Y antigen, NCAM, CEA, T cell epitope, carbohydrate, sialyl-Tn, Globo-H, glycolipid, GD2, GD3 or GM2.

L3 ANSWER 4 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:634026 CAPLUS
DN 141:172878
TI Engineering of glycosylation profile of antibody Fc region to increase Fc receptor binding affinity and effector function for treating cancer
IN Umana, Pablo; Bruenker, Peter; Ferrara, Claudia; Suter, Tobias
PA Glycart Biotechnology Ag, Switz.
SO PCT Int. Appl., 231 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004065540	A2	20040805	WO 2004-IB844	20040122

W: AE, AE, AG, AL, AL, AM, AM, AM, AT, AT, AU, AZ, AZ, BA, BB, BG, BG, BR, BR, BW, BY, BY, BZ, BZ, CA, CH, CN, CN, CO, CO, CR, CR, CU, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EC, EE, EE, EG, ES, ES, FI, FI, GB, GD, GE, GE, GH, GM, HR, HR, HU, HU, ID, IL, IN, IS, JP, JP, KE, KE, KG, KG, KP, KP, KP, KR, KR, KZ, KZ, KZ, LC, LK, LR, LS, LS, LT, LU, LV, MA, MD, MD, MG, MK, MN, MW, MX, MX, MZ, MZ, NA, NI

US 2004241817	A1	20041202	US 2004-761435	20040122

PRAI US 2003-441307P P 20030122
US 2003-491254P P 20030731
US 2003-495142P P 20030815

AB The present invention relates to nucleic acid mols., including fusion constructs, having catalytic activity and the use of same in glycosylation engineering of host cells to generate polypeptides with improved therapeutic properties, including antibodies with increased Fc receptor binding and increased effector function. The engineered proteins or antibodies comprise Golgi localization domain of Golgi resident polypeptide such as .beta.(1,4)-N-acetylglucosaminyltransferase III, .beta.(1,4)-galactosyltransferase, mannosidase II, .beta.(1,2)-N-acetylglucosaminyltransferase I, .beta.(1,2)-N-acetylglucosaminyltransferase II, mannosidase I, .alpha.-mannosidase II, and .alpha.1-6 core fucosyltransferase. The effector function includes Fc-mediated cellular cytotoxicity of NK cells, macrophage, polymorphonuclear cells and monocytes; signaling of apoptosis induction; maturation of dendritic cells; or T cell priming. The engineered antibodies include antibodies or humanized antibodies specific to human neuroblastoma, renal cell carcinoma, colon carcinoma, breast carcinoma, lung carcinoma, ***17*** - ***1A*** antigen, CD20, CD22, CD30, CD40, PSMA, EGFR, PSCA, HLA-DR, MUC1, EpCAM, etc.

L3 ANSWER 5 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:80710 CAPLUS
DN 140:144706
TI Production of recombinant antibodies comprising one common light chain and three different heavy chains for diagnosis and therapy
IN Van Berkel, Patrick Hendrikus Cornelis; Brus, Ronald Hendrik Peter; Bout, Abraham; Logtenberg, Ton
PA Crucell Holland B.V., Neth.
SO PCT Int. Appl., 186 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004009618	A2	20040129	WO 2003-EP7690	20030715
WO 2004009618	A3	20041104		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI EP 2002-77953 A 20020718
US 2002-397066P P 20020718
WO 2003-EP50201 A 20030527

AB The invention provides methods for producing mixts. of antibodies from a single host cell clone. Thereto a nucleic acid sequence encoding a light chain, and nucleic acid sequences encoding different heavy chains are expressed in a recombinant host cell. The antibodies in the mixts. according to the invention suitably comprise identical light chains paired to different heavy chains capable of pairing to the light chain, thereby forming functional antigen binding domains. Antibodies exemplified in the invention include VL and VH of clones K53 (against CD46), UBS-54 (against ***EP*** - ***CAM***), 02-237 (against CD46), B28 (against CD22), II-2 (against CD72) and I-2 (against HLA-DR class II). Such mixts. can be used in a variety of fields.

L3 ANSWER 6 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 1
AN 2004:441672 BIOSIS
DN PREV200400446570
TI Use of the EGP-2/ ***Ep*** - ***CAM*** ***promoter*** for targeted expression of heterologous genes in carcinoma derived cell lines.
AU McLaughlin, Pamela M. J.; Trzpis, Monika; Kroesen, Bart-Jan; Helfrich, Wijnand; Terpstra, Peter; Dokter, Wim H. A.; Ruiters, Marcel H. J.; de Leij, Lou F. M. H.; Harmsen, Martin C. [Reprint Author]
CS Dept Pathol and Lab MedSect Med Biol, Univ Groningen Hosp, Hanzepl 1, NL-9713 GZ, Groningen, Netherlands
m.c.harmsen@med.rug.nl
SO Cancer Gene Therapy, (September 2004) Vol. 11, No. 9, pp. 603-612. print. ISSN: 0929-1903 (ISSN print).
DT Article
LA English
ED Entered STN: 17 Nov 2004
Last Updated on STN: 17 Nov 2004

AB EGP-2, also known as ***Ep*** - ***CAM*** , is expressed at high levels on the surface of most carcinomas and is therefore considered an attractive target for anticancer strategies. To explore the mechanisms regulating the expression of EGP-2, sequences 3.4 kb upstream of the transcription start site were isolated and assayed for their ability to control the expression of the EGP-2 cDNA, the green fluorescent protein, the luciferase reporter gene and the thymidine kinase and cytosine deaminase suicide genes. Expression of these chimeric constructs as assessed in a range of different cell lines was restricted to cell lines expressing EGP-2. In addition, only cells expressing EGP-2 were sensitive for gancyclovir after being transiently transfected with EGP-2 ***promoter*** -driven thymidine kinase. Deletion analyses defined 687 bp upstream as the basic proximal ***promoter*** region, which could confer epithelial-specific expression to the GFP reporter gene in vitro. As these EGP-2 sequences can confer ***promoter*** activity to reporter and suicide genes in an EGP-2 restricted manner, they may be useful for gene therapy of EGP-2 expressing carcinomas.

L3 ANSWER 7 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:472621 CAPLUS
DN 139:51600
TI Chimeric antigen comprising CD36-binding domain for enhancing vaccine immune response
IN Cox, William I.; Alexander, Jeannine P.; Goebel, Scott
PA Aventis Pasteur Limited, Can.
SO PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003050268	A2	20030619	WO 2002-US39885	20021212
WO 2003050268	A3	20040708		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004241652	A1	20041202	US 2002-317821	20021212

PRAI US 2001-341771P P 20011212

AB The invention relates to reagents and methods for enhancing an immune response using CD36 binding region/antigen hybrid polypeptides or polynucleotides encoding the hybrid polypeptides. The antigen is gp100, MART/Melan A, gp75/TRP-1, tyrosinase, NY-ESO-1, melanoma proteoglycan, MAGE-1, MAGE-2, MAGE-3, MAGE-4, MAGE-6, MAGE-12, BAGE, GAGE-1, GAGE-2,
RAGE, N-actylglucosaminyltransferase V, p15, .beta.-catenin, MUM-1, cyclin dependent kinase 4, p21 ras, BCR-abl, p53, p185 HER2/neu, EGF receptor,

CEA antigen, MUC-1, EBNA-1, E7, E6, prostate-specific antigen, prostate specific membrane antigen, KSA, or NY-BR-1.

L3 ANSWER 8 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:472615 CAPLUS
DN 139:30800
TI Streptavidin expressed gene fusions with single-chain antibodies and their use as targeting vehicles for diagnosis and treatment of cancer
IN Goshorn, Stephen Charles; Graves, Scott Stoll; Schultz, Joanne Elaine; Lin, Yukang; Sanderson, James Allen; Reno, John M.; Dearstyne, Erica A.
PA Neorx Corporation, USA
SO PCT Int. Appl., 156 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003050260	A2	20030619	WO 2002-US39429	20021206
WO 2003050260	A3	20041125		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003095977	A1	20030522	US 2001-13173	20011207
US 2003103948	A1	20030605	US 2002-150762	20020517
US 2003143233	A1	20030731	US 2002-244821	20020916
PRAI US 2001-13173	A	20011207		
US 2002-150762	A	20020517		
US 2002-244821	A	20020916		
US 1999-137900P	P	19990607		
US 1999-168976P	P	19991203		
US 2000-589870	A2	20000605		

AB The present invention provides vectors for expressing genomic streptavidin fusion cassettes. In the various embodiments, fusion proteins produced from these vectors are provided. In particular embodiments, fusion proteins comprising a single-chain antibody and genomic streptavidin are provided as are vectors encoding the same. The single-chain antibodies are directed to cell surface antigens, or cell-assocd. stromal or matrix antigens, including, but not limited to, CD20, CD22, CD25, CD45, CD52, CD56, CD57, EGP40 (or EPCAM or KSA), N-CAM, CEA, TAG-72, .gamma.-glutamyl transferase, mucins (MUC1 through MUC7), human .beta.-chorionic gonadotropin, EGF receptor, interleukin-2 receptor, her2/neu, Lewis Y, gangliosides GD2 and GM2, tenascin, sialylated tenascin, somatostatin, activated tumor stromal antigen, or neoangiogenic antigens. Generically, a single-chain Fv/streptavidin (scFvSA) fusion protein is expressed from the genetic fusion of the single-chain antibody of the variable regions to the genomic streptavidin of Streptomyces avidinii. The scFv gene consists of the variable regions of the light and heavy chains sepd. by a DNA linker sequence. The streptavidin coding sequence is joined to the 3'-terminus of the scFv gene, and the two genes are sepd. in-frame by a second DNA linker sequence. The signal sequence from the streptavidin gene is fused at the 5'-terminus of the scFvSA gene to direct expression to the Escherichia coli periplasmic space. The scFvSA gene is under control of the lac ***promoter*** , and the expressed fusion protein is extd. and purified from E. coli and forms a sol. tetramer of .apprx.173,000 mol. wt. Also provided, are methods of using the fusion proteins of the present invention, in the absence and presence of a radiation-sensitizing agent (e.g., Gemcitabine), and in particular, the use of scFvSA fusion proteins as diagnostic markers or as cell-specific targeting agents.

L3 ANSWER 9 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:335307 CAPLUS
DN 138:350812
TI Use of nucleic acid and protein profiling and histology of fixed cells in a single sample in the early diagnosis of disease
IN O'Hara, Shawn Mark; Zweitzig, Daniel; Foulk, Brad
PA Immunivest Corporation, USA
SO PCT Int. Appl., 105 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003035895	A2	20030501	WO 2002-US34570	20021028
WO 2003035895	A3	20040108		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1438419 A2 20040721 EP 2002-795565 20021028
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

PRAI US 2001-330669P	P	20011026		
US 2002-369945P	P	20020404		
WO 2002-US34570	W	20021028		

AB A highly sensitive assay is disclosed which utilizes a method for gene specific primed amplification of mRNA libraries from rare cells and rare transcripts found in blood. The assay allows detection of rare, mRNA (10 copies/cell) found in 1 to 10 cells isolated through immunomagnetic enrichment. The assay is an improvement over multiplex PCR and allows efficient detection of rare coding sequences for circulating carcinoma cells in the blood. The methods are useful in profiling of cells isolated from tissues or body fluids and serves as an adjunct to clin. diagnosis of diverse carcinomas including early stage detection and classification of circulating tumor cells. Monitoring of nucleic acid and protein profiles of cells either in conventional or microarray formats, facilitates management of therapeutic intervention including staging, monitoring response to therapy, confirmation of remission and detection of regression.

L3 ANSWER 10 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:590597 CAPLUS
DN 139:144951
TI Preparation of fusion genes encoding streptavidin and single chain antibody and methods of therapeutic use thereof
IN Goshorn, Stephen Charles; Graves, Scott Stoll; Schultz, Joanne Elaine; Lin, Yukang; Sanderson, James Allen; Reno, John M.; Dearstyne, Erica A.
PA NeoRx Corporation, USA
SO U.S. Pat. Appl. Publ., 89 pp., Cont.-in-part of U.S. Ser. No. 150,762.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003143233	A1	20030731	US 2002-244821	20020916
US 2003095977	A1	20030522	US 2001-13173	20011207
US 2003103948	A1	20030605	US 2002-150762	20020517
WO 2003050260	A2	20030619	WO 2002-US39429	20021206
WO 2003050260	A3	20041125		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-137900P	P	19990607		
US 1999-168976P	P	19991203		
US 2000-589870	A2	20000605		
US 2001-13173	A2	20011207		
US 2002-150762	A2	20020517		
US 2002-244821	A	20020916		

AB The present invention provides vectors for expressing genomic streptavidin fusion cassettes and therapeutic uses. In the various embodiments, fusion proteins produced from these vectors are provided. In particular embodiments, fusion proteins comprising a single chain antibody and genomic streptavidin are provided as are vectors encoding the same. Also provided, are methods of using the fusion proteins of the present invention, in the absence and presence of a radiation-sensitizing agent, and in particular, the use of scFvSA fusion proteins as diagnostic markers or as a cell specific targeting agents.

L3 ANSWER 11 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:435061 CAPLUS
DN 139:21033
TI Vectors expressing soluble form of single chain antibody and streptavidin (scFvSA) fusions and uses thereof as diagnostic markers or as cell specific targeting agents
IN Goshorn, Stephen Charles; Graves, Scott Stoll; Schultz, Joanne Elaine; Lin, Yukang; Sanderson, James Allen; Reno, John M.; Dearstyne, Erica A.
PA NeoRx Corporation, USA
SO U.S. Pat. Appl. Publ., 84 pp., Cont.-in-part of U.S. Ser. No. 13,173.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003103948	A1	20030605	US 2002-150762	20020517
US 2003095977	A1	20030522	US 2001-13173	20011207
US 2003143233	A1	20030731	US 2002-244821	20020916
WO 2003050260	A2	20030619	WO 2002-US39429	20021206
WO 2003050260	A3	20041125		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-137900P P 19990607
US 1999-168976P P 19991203
US 2000-589870 A2 20000605
US 2001-13173 A2 20011207
US 2002-150762 A2 20020517
US 2002-244821 A 20020916

AB The present invention provides vectors for expressing Streptomyces avidinii genomic streptavidin (SA) fusion cassettes. A genomic streptavidin expressed gene fusion is expressed as a sol. protein into the periplasmic space of bacteria and undergoes spontaneous folding. Such expression offers the advantage that the periplasm is a low biotin environment and one need not purify and refold the protein under harsh denaturing conditions that may prove fatal to the polypeptide encoded by a heterologous nucleic acid mol. fused to the genomic streptavidin nucleic acid mol. In the various embodiments, fusion proteins produced from these vectors are provided. In particular embodiments, fusion proteins comprising a single chain antibody and streptavidin (scFvSA) are provided as are vectors encoding the same. The single chain antibodies are directed to cell surface antigens or cell-assocd. stromal or matrix proteins such as CD20, CD45, CD22, CD52, CD56, CD57, EGP40, NCAM, CEA, TAG-72, mucins (MUC1-7), 13HCG, EGF receptor, IL-2 receptor, her2/neu, Lewis Y, GD2, GM2, tenascin, sialylated tenascin, somatostatin, activated tumor stromal antigen or neoangiogenic antigens. Also provided, are methods of using the fusion proteins of the present invention, in the absence and presence of a radiation-sensitizing agent, and in particular, the use of scFvSA fusion proteins as diagnostic markers or as a cell specific targeting agents.

L3 ANSWER 12 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:396269 CAPLUS
DN 138:400405
TI Streptavidin-antibody fusion proteins for diagnosis and specific cell targeting
IN Goshorn, Stephen Charles; Graves, Scott Stoll; Schultz, Joanne Elaine; Lin, Yukang; Sanderson, James Allen; Reno, John M.
PA Neorx Corporation, USA
SO U.S. Pat. Appl. Publ., 72 pp., Cont.-in-part of U.S. Ser. No. 589,870
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003095977	A1	20030522	US 2001-13173	20011207
US 2003103948	A1	20030605	US 2002-150762	20020517
US 2003143233	A1	20030731	US 2002-244821	20020916
WO 2003050260	A2	20030619	WO 2002-US39429	20021206
WO 2003050260	A3	20041125		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-137900P P 19990607
US 1999-168976P P 19991203
US 2000-589870 A2 20000605
US 2001-13173 A2 20011207
US 2002-150762 A2 20020517
US 2002-244821 A 20020916

AB The present invention provides vectors for expressing genomic streptavidin fusion cassettes and fusion protein produced from the vectors. In particular embodiments, fusion proteins comprising a single chain antibody and genomic streptavidin are provided as are vectors encoding the same. Also provided are methods of using the fusion proteins of the present invention, and in particular, the use of scFvSA fusion proteins as diagnostic markers or as a cell specific targeting agents. The single chain antibodies are directed to cell surface antigens or cell-assocd. stromal or matrix protein such as CD20, CD45, CD22, CD52, CD56, CD57, EGP40, NCAM, CEA, TAG-72, mucins (MUC1-7), 13HCG, EGF receptor, IL-2 receptor, her2/neu, Lewis Y, GD2, GM2, tenascin, sialylated tenascin, somatostatin, activated tumor stromal antigen or neoangiogenic antigens.

L3 ANSWER 13 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:704297 CAPLUS
DN 139:346666
TI Cloning and characterisation of a 1.1kb fragment of the carcinoma-associated epithelial cell adhesion molecule ***promoter***
AU Gires, Olivier; Eskofier, Sylvia; Lang, Stephan; Zeidler, Reinhard; Muenz, Markus
CS Clinical Cooperation Group Molecular Oncology, GSF-Research Center for Health and Environment, and Department of Otorhinolaryngology, Ludwig-Maximilians-University, Munich, D-81377, Germany
SO Anticancer Research (2003), 23(4), 3255-3261
CODEN: ANTRD4; ISSN: 0250-7005
PB International Institute of Anticancer Research
DT Journal
LA English

AB The epithelial cell adhesion mol. (EpCAM) is a transmembrane protein assocd. with a variety of carcinomas, where EpCAM is often strongly up-regulated or, as in the case of squamous cell carcinomas, de novo expressed. The mol. mechanisms underlying the transcriptional regulation of EpCAM are poorly understood. So far, a 570bp fragment has been cloned and shown to have specific transcriptional activity, which was neg.-regulated upon the induction of the transcription factor NF-.vkappa.B. In the present study we have cloned a 1100bp fragment of the EpCAM ***promoter*** contg. the 570bp fragment and addnl. 550bp upstream. We demonstrate that both fragments have strong synergistic effects with respect to transcriptional activity in EpCAM-pos. cells. Furthermore, the 1100bp fragment was likewise neg.-regulated upon TNF.alpha. and IFN.alpha. treatment, thus retaining silencer sequences.

RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 14 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:575292 CAPLUS
DN 137:153381
TI Genes overexpressed in prostate disorders as diagnostic and therapeutic targets
IN Hampton, Garret Malcolm; Welsh, John Barnard
PA IRM, LLC, Bermuda
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002059373	A2	20020801	WO 2002-US1615	20020122
WO 2002059373	A3	20040205		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2432991	AA	20020801	CA 2002-2432991	20020122
US 2003013097	A1	20030116	US 2002-54498	20020122
EP 1425413	A2	20040609	EP 2002-709101	20020122

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2004526434	T2	20040902	JP 2002-559855	20020122

PRAI US 2001-263461P P 20010123
US 2001-301639P P 20010628
WO 2002-US1615 W 20020122

AB Disclosed are methods for diagnosing, monitoring the progression of, and treating a prostate disorder based upon genes that are differentially expressed in prostate disorders. Also disclosed are methods for identifying agents useful in the treatment of a prostate disorder, methods for monitoring the efficacy of a treatment for a prostate disorder, methods for inhibiting the proliferation of a prostate cell, and prostate-specific vectors including the ***promoter*** of these genes. A dendrogram of 55 exptl. samples that are grouped according to overall similarity in level of expression of a subset of 3,530 genes that have varied most across the samples is provided. Expression levels of highly ranked genes in normal and malignant prostate tissues are provided. Furthermore, the top 25 or 50 genes (with ref. GenBank accession nos.) overexpressed in prostate malignant tissues or cell lines are identified as the diagnostic markers and therapeutic targets for prostate related disorders. They include genes for hepsin, prostate differentiation factor, alpha-methylacyl-CoA racemase, fatty acid synthase, and prostate specific antigen (alternative splice form 2 and 3). Specifically, the amplification of two marker genes (hepsin and PLAB) are detected at the mRNA level from selected prostate tissues.

L3 ANSWER 15 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:10532 CAPLUS
DN 136:84702
TI Novel ligands for CD28 and CTLA-4 created by shuffling of mammalian B7-1 ligand cDNAs with possible therapeutic use as co-stimulatory molecules
IN Punnonen, Juha; Lazetic, Alexandra L. L.; Leong, Steven R.; Chang, Chia-Chun Jean; Apt, Doris; Gustafsson, Claes
PA Maxygen, Inc., USA
SO PCT Int. Appl., 364 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI WO 2002000717 A2 20020103 WO 2001-US19973 20010622
WO 2002000717 C2 20030206
WO 2002000717 A3 20030821
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
CA 2411828 AA 20020103 CA 2001-2411828 20010622
EP 1360290 A2 20031112 EP 2001-952193 20010622
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR
JP 2004513878 T2 20040513 JP 2002-505839 20010622
US 2003138881 A1 20030724 US 2001-32214 20011220
WO 2004029197 A2 20040408 WO 2002-US19898 20020621
WO 2004029197 A3 20041028
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1497426 A2 20050119 EP 2002-807658 20020621
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRAI US 2000-213946P P 20000623
US 2000-241245P P 20001017
US 2001-888324 A2 20010622
WO 2001-US19973 W 20010622
US 2001-32214 A2 20011220
WO 2002-US19898 W 20020621
AB The invention provides polynucleotides and polypeptides encoded therefrom having advantageous properties, including an ability of the polypeptides to preferentially bind a CD28 or CTLA-4 receptor at a level greater or less than the ability of human B7-1 to bind CD28 or CTLA-4, or to induce or inhibit altered level of T cell proliferation response greater compared to that generated by human B7-1. The polypeptides and polynucleotides of the invention are useful in therapeutic and prophylactic treatment methods, gene therapy applications, and vaccines. Novel ligands were generated by shuffling of sequences from cDNAs for B7-1 ligands from human, rhesus monkey, baboon, orangutan, cow, cat and rabbit. Ligands were screened for using a FACS assay. CDNA libraries were introduced into animal cells that were then screened for their ability to bind a labeled CD28 or CTLA-4 using FACS. Clones were screened for their preferential binding of CD28 vs. CTLA-4. Candidate clones were then tested for their ability to stimulate T cell proliferation.

L3 ANSWER 16 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 2
AN 2002:523713 BIOSIS
DN PREV200200523713
TI Murine spermatogonial stem cells: Targeted transgene expression and purification in an active state.
AU Giuili, Galicia; Tomljenovic, Andrea; Labrecque, Nathalie; Oulad-Abdelghani, Mustapha; Rassoulzadegan, Minoo; Cuzin, Francois [Reprint author]
CS Unite 470 de l'INSERM, Universite de Nice, F-06108, Nice Cedex 2, France fcuzin@unice.fr
SO EMBO Reports, (August, 2002) Vol. 3, No. 8, pp. 753-759. print. ISSN: 1469-221X.
DT Article
LA English
ED Entered STN: 9 Oct 2002
Last Updated on STN: 9 Oct 2002
AB A 400 bp fragment of the spermatogonia-specific Stra8 locus was sufficient to direct gene expression to the germinal stem cells in transgenic mice. A fractionation procedure was devised, based on immunomagnetic sorting of cells in which the ***promoter*** drives the expression of a surface functionally neutral protein tag. The purified cells expressed the known molecular markers of spermatogonia Rbm, cyclin A2 and ***EP*** - ***Cam*** , and the beta1- and alpha6-integrins characteristic of the stem cell fraction. A 700-fold enrichment in stem cells was determined by the ability of the purified fractions to re-establish spermatogenesis in germ cell-depleted recipient testes.

L3 ANSWER 17 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:816487 CAPLUS
DN 135:356752
TI Epitope synchronization in antigen presenting cells
IN Simard, John J. L.; Diamond, David C.; Lei, Xiang-Dong
PA CTL Immunotherapies Corp., USA
SO PCT Int. Appl., 131 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 7
PATENT NO. KIND DATE APPLICATION NO. DATE
PI WO 2001082963 A2 20011108 WO 2001-US13806 20010427
WO 2001082963 A3 20020411
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
CA 2405363 AA 20011108 CA 2001-2405363 20010427
EP 1276896 A2 20030122 EP 2001-930922 20010427
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
JP 2003535824 T2 20031202 JP 2001-579836 20010427
PRAI US 2000-560465 A 20000428
US 2000-561074 A 20000428
US 2000-561571 A 20000428
US 2000-561572 A 20000428
WO 2001-US13806 W 20010427
AB Disclosed herein are vaccines and methods for inducing an immune response against cancer cells and cells infected with intracellular parasites. Vaccines having housekeeping epitopes are disclosed. The housekeeping epitope is formed by housekeeping proteasomes in peripheral cells, but not by professional antigen presenting cells. A vaccine contg. a housekeeping epitope that is derived from an antigen assocd. with a peripheral target cell can thus direct an immune response against the target cell. Methods of treatment are also disclosed, which involve administering a vaccine having a housekeeping epitope.

L3 ANSWER 18 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:338579 CAPLUS
DN 134:365705
TI Antibody diversity generation
IN Karrer, Erik; Bass, Steven H.; Whalen, Robert; Patten, Phillip A.
PA Maxygen, Inc., USA
SO PCT Int. Appl., 109 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE
PI WO 2001032712 A2 20010510 WO 2000-US30247 20001101
WO 2001032712 A3 20020321
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
EP 1230269 A2 20020814 EP 2000-976844 20001101
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRAI US 1999-163370P P 19991103
US 2000-176002P P 20000112
WO 2000-US30247 W 20001101
AB Methods for improving antibodies by a variety of DNA diversification and selection procedures are provided. Improvements include increases in affinity, alterations in specificity and effector function, as well as reduced antigenicity, e.g. humanization. Libraries of recombinant antibody sequences are provided, as are cells expressing members of such libraries. Novel phage display vectors are provided. Methods for the coevolution of an antibody and its cognate antigen are provided. Coevolution is used to evolve HIV envelope proteins with increased antigenicity and broadly neutralizing antibodies that interact therewith. Methods of improving antibodies for use in the detection of biol. warfare agents are provided.

L3 ANSWER 19 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 3
AN 2001:300365 BIOSIS
DN PREV200100300365
TI The ***epithelial*** ***glycoprotein*** ***2*** (EGP-2) ***promoter*** -driven epithelial-specific expression of EGP-2 in transgenic mice: A new model to study carcinoma-directed immunotherapy.
AU McLaughlin, Pamela M. J.; Harmsen, Martin C.; Dokter, Wim H. A.; Kroesen, Bart-Jan; van der Molen, Henk; Brinker, Marja G. L.; Hollema, Harry; Ruiters, Marcel H. J.; Buys, Charles H. C. M.; de Leij, Lou F. M. H. [Reprint author]
CS Department of Pathology and Laboratory Medicine, Section Medical Biology,

University Hospital Groningen, Hanzeplein 1, 9713 GZ, Groningen, Netherlands
l.f.m.h.de.leij@med.rug.nl
SO Cancer Research, (May 15, 2001) Vol. 61, No. 10, pp. 4105-4111. print.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
LA English
ED Entered STN: 20 Jun 2001
Last Updated on STN: 19 Feb 2002
AB The human pancarcinoma-associated ***epithelial*** ***glycoprotein*** - ***2*** (EGP-2), a Mr 38,000 transmembrane antigen also known as ***17*** - ***1A*** or ***Ep*** - ***CAM*** , is commonly used for targeted immunotherapy of carcinomas because it is strongly expressed by most carcinomas. EGP-2 is, however, also expressed in most normal epithelia. To evaluate anti-EGP-2-directed treatment-associated effects on tumors and on EGP-2-positive normal tissue, we generated EGP-2-expressing transgenic mice. A 55-kb DNA fragment consisting of the 14-kb genomic coding sequence of the human EGP-2 gene with apprx10-kb-upstream and apprx31-kb-downstream sequences was isolated and used to direct EGP-2 expression in an epithelium-specific manner. In the EGP-2 transgenic mice, EGP-2 appeared to be specifically expressed in all of those epithelial tissues that also express EGP-2 in humans, whereas all of the other tissues were negative. The specific in vivo localization of the i.v. administered anti-EGP-2 monoclonal antibody MOC31 was studied in EGP-2-positive and -negative tumors induced s.c. in this EGP-2 transgenic mouse model. Immunohistochemical analysis showed specific localization of MOC31 in the EGP-2-positive tumors but not in the EGP-2-negative tumors. No anti-EGP-2 monoclonal antibody localization was observed in any of the EGP-2-positive normal mouse tissues, which indicated a limited in vivo accessibility. In conclusion, an EGP-2 transgenic mouse model has been generated that expresses the EGP-2 antigen as in humans and, therefore, can serve as a model to evaluate the efficacy and safety of a variety of anti-EGP-2-based immunotherapeutic modalities in both tumors and normal tissue.

L3 ANSWER 20 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:881321 CAPLUS
DN 134:38630
TI Streptavidin expressed gene fusions forming tetrameric complexes with therapeutic implications for adenocarcinomas and hematol. malignancies
IN Goshorn, Stephen Charles; Graves, Scott Stoll; Schultz, Joanne Elaine; Lin, Yukang; Sanderson, James Allen; Reno, John M.
PA Neorx Corp., USA
SO PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000075333	A1	20001214	WO 2000-US15595	20000605
WO 2000075333	C2	20020620		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2376192	AA	20001214	CA 2000-2376192	20000605
EP 1190061	A1	20020327	EP 2000-941246	20000605
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2003501096	T2	20030114	JP 2001-502595	20000605
PRAI US 1999-137900P	P	19990607		
US 1999-168976P	P	19991203		
WO 2000-US15595	W	20000605		

AB The present invention provides vectors for expressing genomic streptavidin fusion cassettes which include inducible promoters and various linkers and signal sequences. In the various embodiments, fusion proteins produced from these vectors are provided. In particular embodiments, fusion proteins comprising a single chain antibody (huNR-LU-10) and genomic streptavidin are provided as are vectors encoding the same. Also provided, are methods of using the fusion proteins of the present invention, and in particular, the use of scFvSA fusion proteins involving B9E9 as diagnostic markers or as a cell specific targeting agents. In addn. tetravalent antibodies that contact a fusion protin forming a tetrametric complex which may comprise a tumor cell surface-assocd. protein and a streptavidin portion capable of binding biotin and a biotinylated radionuclide contg. compd. A immunoreactivity assay is described in addn. to monitoring of blood clearance and tumor uptake of fusion proteins. Some adenocarcinomas and hematol. malignancies such as non-Hodgkin's lymphoma may be treated with these fusio-protein expressing vectors. This system offers the expression of a genomic streptavidin gene fusion as a sol. protein into the periplasmic space of Escherichia coli where it undergoes spontaneous folding. This expression offers efficient protein folding where one does not need to purify and refold the protein expressed.
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 21 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:824304 CAPLUS
DN 134:16539
TI Antibodies
IN Hoogenboom, Hendricus Renerus Jacobus Mattheus; Reurs, Anneke; Beiboer, Sigrid Herma Wilma
PA Oxford Biomedica (UK) Limited, UK
SO PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000069914	A2	20001123	WO 2000-GB1910	20000518
WO 2000069914	A3	20010405		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRAI GB 1999-11569	A	19990518		

AB Human antibodies that recognize the epithelial glycoprotein antigen (EGP-2) are disclosed. The antibodies have a human light chain variable region and a human heavy chain variable region. Fragments of the antibodies and pharmaceutical compns. comprising the antibodies and their in vitro and in vivo applications in diagnosis and immunotherapy are also disclosed.

L3 ANSWER 22 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:402017 CAPLUS
DN 133:54574
TI Recombinant vectors expressing multiple costimulatory molecules, host cell infection, and uses in immunogenic applications
IN Schlom, Jeffrey; Hodge, James; Panicali, Dennis
PA United States Dept. of Health and Human Services, USA; Therion Biologics Corporation
SO PCT Int. Appl., 188 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000034494	A1	20000615	WO 1999-US26866	19991112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2354024	AA	20000615	CA 1999-2354024	19991112
EP 1137792	A1	20011004	EP 1999-958951	19991112
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002531133	T2	20020924	JP 2000-586927	19991112
AU 774076	B2	20040617	AU 2000-16218	19991112
US 2004019195	A1	20040129	US 2003-406317	20030404
PRAI US 1998-111582P	P	19981209		
WO 1999-US26866	W	19991112		
US 2001-856988	A3	20010924		

AB The present invention provides recombinant vectors encoding and expressing at least three or more costimulatory mols and host cells infected by the vector. The recombinant vector may addnl. contain a gene encoding one or more target antigens or immunol. epitope as well as cytokine, chemokine, or Flt-3L. A method of making a recombinant poxvirus, of enhancing an immune response of an individual by administering a recombinant vector, and of treating or preventing a disease by activating a T lymphocyte, are also presented. Further describes are a method of making a progenitor dendritic cell or dendritic cell, of assessing the efficacy of a vaccine against a target antigen, and of screening for novel immunogenic peptides. The synergistic effect of these costimulatory mols. on the enhanced activation of T cells was demonstrated. The degree of T-cell activation using recombinant vectors contg. genes encoding three costimulatory mols. was far greater than the sum of recombinant vector constructs contg. one costimulatory mol. and greater than the use of two costimulatory mols. Results employing the triple costimulatory vectors were most dramatic under conditions of either low levels of first signal or low stimulator to T-cell ratios. This phenomenon was obsd. with both isolated CD4+ and CD8+ T cells. The recombinant vectors of the present invention are useful as immunogenes and vaccines against cancer and pathogenic micro-organisms, and in providing host cells, including dendritic cells and splenocytes with enhanced antigen-presenting functions.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 23 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:240985 CAPLUS
DN 132:292701
TI Novel methods for therapeutic vaccination
IN Steinaa, Lucilla; Mouritsen, Soren; Nielsen, Klaus Gregorious; Haaning, Jesper; Leach, Dana; Dalum, Iben; Gautam, Anand; Birk, Peter; Karlsson, Gunilla
PA M & E Biotech A/S, Den.
SO PCT Int. Appl., 220 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000020027	A2	20000413	WO 1999-DK525	19991005
WO 2000020027	A3	20001012		
W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2345817	AA	20000413	CA 1999-2345817	19991005
AU 9958510	A1	20000426	AU 1999-58510	19991005
AU 751709	B2	20020822		
EP 1117421	A2	20010725	EP 1999-945967	19991005
EP 1117421	B1	20040616		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, MC, IE, SI, LT, LV, FI, RO				
TR 200100936	T2	20010821	TR 2001-200100936	19991005
JP 2002526419	T2	20020820	JP 2000-573386	19991005
EE 200100203	A	20021015	EE 2001-203	19991005
NZ 511055	A	20031031	NZ 1999-511055	19991005
AT 269100	E	20040715	AT 1999-945967	19991005
NO 2001001586	A	20010531	NO 2001-1586	20010328
ZA 2001002603	A	20020930	ZA 2001-2603	20010329
HR 2001000319	A1	20020630	HR 2001-319	20010504
US 2004141958	A1	20040722	US 2003-441779	20030519
PRAI DK 1998-1261	A	19981005		
US 1998-105011P	P	19981020		
US 1999-413186	A1	19991005		
WO 1999-DK525	W	19991005		

AB A method is disclosed for inducing cell-mediated immunity against cellular antigens. More specifically, the invention provides for a method for inducing cytotoxic T-lymphocyte immunity against weak antigens, notably self-proteins. The method entails that antigen presenting cells are induced to present at least one CTL epitope of the weak antigen and at the same time presenting at least one foreign T-helper lymphocyte epitope. In a preferred embodiment, the antigen is a cancer specific antigen, e.g. prostate specific membrane antigen (PSM), Her2, or FGF8b. The method can be exercised by using traditional polypeptide vaccination, but also by using live attenuated vaccines or nucleic acid vaccination. The invention furthermore provides immunogenic analogs of PSM, Her2 and FGF8b, as well as nucleic acid mols. encoding these analogs. Also vectors and transformed cells are disclosed. The invention also provides for a method for identification of immunogenic analogs of weak or non-immunogenic antigens.

L3 ANSWER 24 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:795994 CAPLUS
DN 132:31744
TI Gene probes used for genetic profiling in healthcare screening and planning
IN Roberts, Gareth Wyn
PA Genostic Pharma Ltd., UK
SO PCT Int. Appl., 745 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9964627	A2	19991216	WO 1999-GB1780	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRAI GB 1998-12099	A	19980606		
GB 1998-13291	A	19980620		
GB 1998-13611	A	19980624		
GB 1998-13835	A	19980627		
GB 1998-14110	A	19980701		
GB 1998-14580	A	19980707		
GB 1998-15438	A	19980716		
GB 1998-15574	A	19980718		
GB 1998-15576	A	19980718		
GB 1998-16085	A	19980724		
GB 1998-16086	A	19980724		
GB 1998-16921	A	19980805		
GB 1998-17097	A	19980807		
GB 1998-17200	A	19980808		
GB 1998-17632	A	19980814		
GB 1998-17943	A	19980819		

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L3 ANSWER 25 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:795993 CAPLUS
DN 132:31743
TI Gene probes used for genetic profiling in healthcare screening and planning
IN Roberts, Gareth Wyn
PA Genostic Pharma Limited, UK
SO PCT Int. Appl., 149 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9964626	A2	19991216	WO 1999-GB1779	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2330929	AA	19991216	CA 1999-2330929	19990604
AU 9941586	A1	19991230	AU 1999-41586	19990604
AU 766544	B2	20031016		
AU 9941587	A1	19991230	AU 1999-41587	19990604
GB 2339200	A1	20000119	GB 1999-12914	19990604
GB 2339200	B2	20010912		
EP 1084273	A1	20010321	EP 1999-925207	19990604
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2003528564	T2	20030930	JP 2000-553616	19990604
US 2003198970	A1	20031023	US 2002-206568	20020729
PRAI GB 1998-12098	A	19980606		
GB 1998-28289	A	19981223		
GB 1998-16086	A	19980724		
GB 1998-16921	A	19980805		
GB 1998-17097	A	19980807		
GB 1998-17200	A	19980808		
GB 1998-17632	A	19980814		
GB 1998-17943	A	19980819		
US 1999-325123	B1	19990603		
WO 1999-GB1779	W	19990604		

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA

sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

L3 ANSWER 26 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:691109 CAPLUS
DN 131:335805
TI Glycosylation engineering of antibodies for improving antibody-dependent cellular cytotoxicity
IN Umana, Pablo; Jean-Mairet, Joel; Bailey, James E.
PA Switz.
SO PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9954342	A1	19991028	WO 1999-US8711	19990420
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9936578	A1	19991108	AU 1999-36578	19990420
EP 1071700	A1	20010131	EP 1999-918731	19990420
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002512014	T2	20020423	JP 2000-544680	19990420
US 6602684	B1	20030805	US 1999-294584	19990420
US 2004072290	A1	20040415	US 2003-437388	20030514
PRAI US 1998-82581P	P	19980420		
US 1999-294584	A1	19990420		
WO 1999-US8711	W	19990420		

AB The present invention relates to the field of glycosylation engineering of proteins. More particularly, the present invention is directed to the glycosylation engineering of proteins to provide proteins with improved therapeutic properties, e.g., antibodies, antibody fragments, or a fusion protein that includes a region equiv. to the Fc region of an Ig, with enhanced Fc-mediated cellular cytotoxicity. The antibodies or fusion proteins with enhanced Fc-mediated cellular cytotoxicity are expressed in host cells engineered to also express a glycoprotein-modifying glycosyl transferase, e.g. .beta.(1,4)-N-acetylglucosaminyltransferase III or V, .beta.(1,4)-N-galactosyltransferase, and mannosidase II.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 27 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:173463 CAPLUS
DN 128:304704
TI A -308 deletion of the tomato LAP promoters is able to direct flower-specific and MeJA-induced expression in transgenic plants
AU Ruiz-Rivero, Omar J.; Prat, Salome
CS Dpto. de Genetica Molecular, Centro de Investigacion y Desarrollo-C.S.I. C., Barcelona, 08034, Spain
SO Plant Molecular Biology (1998), 36(5), 639-648
CODEN: PMBIDB; ISSN: 0167-4412
PB Kluwer Academic Publishers
DT Journal
LA English
AB Tomato and potato leucine aminopeptidase (LAP) mRNAs are induced in response to mech. wounding and the wound signal mols., ABA and jasmonic acid. Here, we report the isolation of two LAP genes, LAP17.1A and LAP17.2, from tomato. Functional anal. in transgenic tomato and potato plants show that fusions of the corresponding 5' non-coding regions to the gusA gene are constitutively expressed in flowers and induced in leaves upon wounding or by treatment with Me jasmonate (MeJA). Comparison of the 5' non-coding regions of the two genes revealed a region from -317 to -3 relative to the ATG, which is strongly conserved in both promoters. This 0.3 kb proximal ***promoter*** fragment is sufficient to direct flower-specific and MeJA-inducible GUS activity in transgenic potato plants, and thus contains a MeJA-responsive element that mediates induction by MeJA. Dimeric TGACG motifs or G-box elements similar to those found in other MeJA-inducible genes are not obsd. in this region, which suggests that a different DNA sequence is involved in MeJA induction of the LAP genes.
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 28 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 4
AN 1998:436540 BIOSIS
DN PREV199800436540
TI The impact of antigen density and antibody affinity on antibody-dependent cellular cytotoxicity: Relevance for immunotherapy of carcinomas.
AU Velders, M. P.; Van Rhijn, C. M.; Oskam, E.; Fleuren, G. J.; Warnaar, S. O.; Litvinov, S. V. [Reprint author]
CS Dep. Pathol., Leiden Univ. Hosp. Build. 1, L1-Q, PO Box 9600, 2300 RC Leiden, Netherlands
SO British Journal of Cancer, (Aug., 1998) Vol. 78, No. 4, pp. 478-483. print.
CODEN: BJCAAI. ISSN: 0007-0920.
DT Article
LA English
ED Entered STN: 7 Oct 1998
Last Updated on STN: 7 Oct 1998
AB Antibody-dependent cellular cytotoxicity (ADCC) is considered to be the major mechanism through which tumour cells, upon treatment with anti-tumour MAbs, are eliminated in vivo. However, the relative importance of various parameters that influence the efficacy of ADCC is unclear. Here we present in vitro data on the impact of MAb affinity and antigen density on ADCC, as obtained by comparison of two MAbs against the tumour-associated antigen ***Ep*** - ***CAM*** . The low-affinity MAb ***17*** - ***1A*** (Ka = 5 X 107 M-1) currently used for therapy, and the high-affinity MAb 323/A3 (Ka = 2 X 109 m-1), were compared in ADCC experiments against murine and human tumour target cells transfected with the ***Ep*** - ***CAM*** cDNA under the control of an inducible ***promoter*** to enable regulation of the target antigen expression levels. Data obtained from these studies revealed that the high-affinity MAb, in contrast to the low-affinity MAb, could mediate killing of tumour cells with low antigen expression levels. Even at comparable MAb-binding levels, ADCC mediated by the high-affinity MAb was more effective. The kinetics of ADCC was also found to be determined by the level of antigen expression, and by the affinity and the concentration of the MAb used. The efficacy of ADCC with both low- and high-affinity MAbs further depended on adhesive interactions between effector and target cells mediated by CD18. However, at every given MAb concentration these interactions were of less importance for the high-affinity MAb than for the low-affinity MAb. As heterogeneity of a target antigen expression is a common feature of all tumours, and some tumour cells express very low levels of the antigen, the use of high-affinity MAbs in immunotherapy may significantly improve the clinical results obtained to the present date in the treatment of minimal residual disease.

L3 ANSWER 29 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1997:97727 CAPLUS
DN 126:156420
TI Prophylactic and therapeutic vector vaccination using expression constructs for individual epitopes of antigens
IN Weiner, David B.; Williams, William V.; Wang, Bin
PA Wistar Institute, USA; Trustees of the University of Pennsylvania
SO U.S., 50 pp., Cont.-in-part of U.S. Ser. No. 29,336, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5593972	A	19970114	US 1993-125012	19930921
ZA 9400493	A	19950103	ZA 1994-493	19940125
CA 2153593	AA	19940804	CA 1994-2153593	19940126
WO 9416737	A1	19940804	WO 1994-US899	19940126
W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, KZ, LK, LU, LV, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SK, UA, US, US, US, US, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9462320	A1	19940815	AU 1994-62320	19940126
AU 675702	B2	19970213		
EP 681483	A1	19951115	EP 1994-909492	19940126
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
HU 73099	A2	19960628	HU 1995-2229	19940126
HU 219767	B	20010730		
JP 08509694	T2	19961015	JP 1994-517285	19940126
EP 1473369	A2	20041103	EP 2004-75092	19940126
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
US 6348449	B1	20020219	US 1994-357398	19941216
US 5830876	A	19981103	US 1995-453349	19950530
US 5817637	A	19981006	US 1997-783818	19970113
US 6468982	B1	20021022	US 1997-880576	19970623
US 5981505	A	19991109	US 1997-979385	19971126
PRAI US 1993-8342	B2	19930126		
US 1993-29336	B2	19930311		
US 1993-93235	A	19930715		
US 1993-124962	A	19930921		
US 1993-125012	A	19930921		
EP 1994-909492	A3	19940126		
WO 1994-US899	W	19940126		
US 1995-495684	B1	19950828		
US 1997-783818	A1	19970113		

AB Methods of prophylactic and therapeutic immunization against infection, hyperproliferative and autoimmune diseases are disclosed. An expression construct directing the synthesis of one or more epitopes, or analogs of

epitopes, of an antigen is introduced into cells of an individual. The epitope is identical or substantially similar to an epitope of a pathogen antigen, a hyperproliferative cell assocd. protein or a protein assocd. with autoimmune disease resp. Methods of immunizing against HIV are described. Succesful induction of immunity to HIV1 in mice by injection with an expression vector for the HIV-1 gene env.

L3 ANSWER 30 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1996:278128 CAPLUS
DN 124:307777
TI Dynamic monitoring and quantification of gene expression in single, living cells: a molecular basis for secretory cell heterogeneity
AU Castano, Justo P.; Kineman, Rhonda D.; Frawley, L. Stephen
CS Dep. Cell Biology Anat., Med. Univ. South Carolina, Charleston, SC, 29425, USA
SO Molecular Endocrinology (1996), 10(5), 599-606
CODEN: MOENEN; ISSN: 0888-8809
PB Endocrine Society
DT Journal
LA English
AB Progress in understanding the dynamics of gene expression has been hampered by lack of a strategy for continuously monitoring this process within normal, living cells. Here, the authors employed a modifn. of conventional luciferase technol. to make single and repeated real-time measurements of PRL gene expression from individual, living lactotropes from nursing rats. Cells were individually transfected by microinjection with a PRL ***promoter*** /luciferase reporter construct. Levels of PRL gene transcription were quantified by photonic imaging in the same cells before and after 24 h of culture in the presence or absence of the dopamine agonist bromocryptine or ***EGF*** , ***2*** well known regulators of PRL gene transcription. These cells were found to be remarkably heterogeneous with respect to basal PRL gene expression and that the degree of activity within a single cell could fluctuate greatly over time under basal culture conditions. Treatment with bromocryptine or EGF induced predictable and reversible changes in the av. responses obsd., yet individual cells displayed marked differences in responses to these agents. These findings demonstrate the utility of this paradigm for monitoring dynamics of gene expression within normal, living cells of any type. Moreover, they provide a mol. basis for the secretory heterogeneity and plasticity that have come to be known as hallmarks of lactotrope cell function.

L3 ANSWER 31 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1995:319826 CAPLUS
DN 122:98808
TI Cloning and expression of human .beta.2-microglobulin cDNA and the construction of fusion proteins between antigenic epitopes and .beta.2-microglobulin
IN Edwards, Richard Mark; Hunter, Michael George
PA British Bio-Technology Ltd., UK
SO PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9424290	A1	19941027	WO 1994-GB755	19940408
W: AU, BR, CA, CN, CZ, DE, FI, GB, HU, JP, KR, NO, NZ, PL, RU, UA, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9464353	A1	19941108	AU 1994-64353	19940408
EP 693125	A1	19960124	EP 1994-912040	19940408
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE				
US 2002123108	A1	20020905	US 1995-532549	19951201
PRAI GB 1993-7371	A	19930408		
WO 1994-GB755	W	19940408		

AB A method is described for the cloning and expression of human .beta.2-microglobulin (B2M) cDNA in vector host cells which allows the construction of B2M fusion proteins with antigenic sequences from various etiol. agents or tumors. Preferred antigenic sequences are derived from the third variable domain (V3 loop) of an envelope protein of a lentivirus. These fusion proteins can be used as prophylactic or immunotherapeutic vaccines to induce neutralizing antibody responses. Thus, B2M cDNA was inserted into the pHILD1 expression vector for expression in the Pichia pastoris system. The expression vector includes an AOX ***promoter*** sequence and an .alpha.-factor or Pho1 leader sequence to obtain secretion of the fusion protein from the yeast cells. Within the Pichia pastoris expression system, the B2M gene was fused at its 5' end to the Sendai virus epitope (FAPGNYPAL-GGGGG, where the pentaglycine is a short linker) or to the influenza A virus nucleoprotein epitope (GILGFVFTL-GGGGGGSSS). Prodn. levels from strains with the .alpha.-factor leader sequence were .apprx.150 mg/L. The hybrid Sendai-B2M product was shown to induce Sendai nucleoprotein-specific cytotoxic T-lymphocytes.

L3 ANSWER 32 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1994:623662 CAPLUS
DN 121:223662
TI Genetic transformation of animal cells using agents that stimulate DNA uptake or gene expression or the inflammatory response
IN Weiner, David B.; Williams, William V.; Wang, Bin; Coney, Leslie R.; Merva, Michael J.; Zurawski, Vincent R., Jr.
PA USA
SO PCT Int. Appl., 135 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9416737	A1	19940804	WO 1994-US899	19940126
W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, KZ, LK, LU, LV, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SK, UA, US, US, US, US, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5593972	A	19970114	US 1993-125012	19930921
ZA 9400493	A	19950103	ZA 1994-493	19940125
CA 2153593	AA	19940804	CA 1994-2153593	19940126
AU 9462320	A1	19940815	AU 1994-62320	19940126
AU 675702	B2	19970213		
EP 681483	A1	19951115	EP 1994-909492	19940126
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 08509694	T2	19961015	JP 1994-517285	19940126
RU 2174845	C2	20011020	RU 1995-117922	19940126
US 5981505	A	19991109	US 1997-979385	19971126
PRAI US 1993-8342	A	19930126		
US 1993-29336	A	19930311		
US 1993-93235	A	19930715		
US 1993-124962	A	19930921		
US 1993-125012	A	19930921		
WO 1994-US899	W	19940126		
US 1995-495684	B1	19950828		

AB Methods of introducing nucleic acids into cells of an individual using agents that stimulate nucleic acid uptake or expression or the inflammatory response are described. The method avoids the use of viral or retroviral particles. The transforming nucleic acid encodes an antigenic peptide and so may be useful in therapeutics or prophylaxis. Methods of prophylactically and therapeutically immunizing an individual against HIV without the use of retroviral proteins or particles are disclosed. Expression cassettes for manuf. of antigens of HIV-1 in animal cells were constructed by std. methods. These were used to transform tumor cell lines not normally recognized by a mouse host. Mice injected with these transformed cells mounted a strong cytotoxic response that completely eliminated tumors that would normally kill the animal in 12 wk. Injection of mice with an expression vector carrying an expression cassette for gp160 in combination with bupivacaine to stimulate inflammation and cell proliferation resulted in a strong immune response to gp160. The response was stronger than from mice injected with gp160 or injected with the expression vector without the use of bipuvacaine.

L3 ANSWER 33 OF 37 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 5
AN 95036827 EMBASE
DN 1995036827
TI Studies on ***17*** - ***1A*** antigen gene regulation in nonexpressing A549 and A431 cells, as compared to expressing pancreatic carcinoma (Capan 2) cells, reveal a complex mechanism of repression of this gene.
AU Siemieniako B.; Wiland E.; Trzeciak W.H.
CS Inst. of Biochemistry/Biotechnology, University of Agriculture, Wolynska 35,60-637 Poznan, Poland
SO Cell Biology International, (1994) 18/11 (1009-1017).
ISSN: 1065-6995 CODEN: CBIIEV
CY United Kingdom
DT Journal; Article
FS 016 Cancer
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
AB Elements controlling high expression of the ***17*** - ***1A*** antigen gene in pancreatic carcinoma cells (Capan 2) reside within the two regions: proximal (-193 to +3) and distal (-877 to -518). We demonstrate here that some factors present in nuclear extracts from nonexpressing cells bind specifically to the control elements, important for gene expression. Our results suggest that nonexpressing cells may either lack at least one of the factors necessary for activation or may contain their modified forms. A major difference between expressing and nonexpressing cells was found in the region containing core enhancer sequence. Moreover, nonexpressing cells display a complex pattern of DNA-protein interactions in this region, suggesting that these cells contain factors acting negatively mainly on the enhancer sequence. Our results however, indicate that the mechanism of repression is much more complicated than expected.

L3 ANSWER 34 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 6
AN 1993:207235 BIOSIS
DN PREV199395108460
TI Retroposition in a family of carcinoma-associated antigen genes.
AU Linnenbach, Alban J. [Reprint author]; Seng, Beth A.; Wu, Shuang; Robbins, Shira; Scollon, Maureen; Pyrc, Jania J.; Druck, Teresa; Huebner, Kay
CS Wistar Inst., 3601 Spruce St., Philadelphia, PA 19104, USA
SO Molecular and Cellular Biology, (1993) Vol. 13, No. 3, pp. 1507-1515.

CODEN: MCEBD4. ISSN: 0270-7306.
DT Article
LA English
OS Genbank-M93029; Genbank-M93030; Genbank-M93031; Genbank-M93032; Genbank-M93033; Genbank-M93034; Genbank-M93035; Genbank-M93036; Genbank-X13425
ED Entered STN: 23 Apr 1993
Last Updated on STN: 9 Jun 1993
AB The gene encoding the carcinoma-associated antigen defined by the monoclonal antibody GA733 is a member of a family of at least two type I membrane proteins. This study describes the mechanism of evolution of the GA733-1 and ***GA733*** - ***2*** genes. A full-length cDNA clone for GA733-1 was obtained by screening a human placental library with a genomic DNA probe. Comparative analysis of the cDNA sequence with the previously determined genomic sequence confirmed that GA733-1 is an intronless gene. The ***GA733*** - ***2*** gene encoding the monoclonal antibody-defined antigen was molecularly cloned with a cDNA probe and partially sequenced. Comparison of ***GA733*** - ***2*** gene sequences with the previously established cDNA sequence revealed that this gene consists of nine exons. The putative ***promoter*** regions of the GA733-1 and ***GA733*** - ***2*** genes are unrelated. These findings suggest that the GA733-1 gene was formed by the retroposition of the ***GA733*** - ***2*** gene via an mRNA intermediate. Prior to retroposition, the ***GA733*** - ***2*** gene had been affected by exon shuffling. Analysis of ***GA733*** - ***2*** exons revealed that many delineate structural motifs. The GA733-1 retroposon was localized either to chromosome region 1p32-1p31 or to 1p13-1q12, and the ***GA733*** - ***2*** founder gene was localized to chromosome 4q.

L3 ANSWER 35 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 7
AN 1992:476438 BIOSIS
DN PREV199294107813; BA94:107813
TI NUCLEAR PROTEINS FROM CAPAN-2 CELL LINE FORM SPECIFIC COMPLEXES WITH THE
17-1 A ANTIGEN GENE ***PROMOTER*** .
AU SIEMIENIAKO B [Reprint author]; WILAND E
CS INST HUMAN GENETICS, POLISH ACADEMY SCI, STRZESZYNSKA 32, 60-479 POZNAN,
POLAND
SO Biochemical and Biophysical Research Communications, (1992) Vol. 186, No. 3, pp. 1353-1361.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 27 Oct 1992
Last Updated on STN: 27 Oct 1992
AB To determine the location of sites important for the function of the ***17*** - ***1A*** antigen gene ***promoter*** and to characterize the protein factors binding to these sites, fragments of the ***promoter*** region were analysed by gel retardation assay with nuclear extracts from Capan 2 cell line. At least two separate regions, which specifically bind nuclear proteins were identified within the 5'flanking region of the ***17*** - ***1A*** antigen gene. These regions have been located between nucleotides -877 to -518 (distal region) and -193 to +3 (proximal region) and presumably participate in regulation of expression of the ***17*** - ***1A*** antigen gene.

L3 ANSWER 36 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 1992:134089 BIOSIS
DN PREV199242061789; BR42:61789
TI HUMAN ***17*** - ***1A*** NEOANTIGEN GENE ***PROMOTER*** .
AU WOJCIEROWSKI J [Reprint author]; POLUHA D; ZIELEWICZ J
CS DEP MED GENETICS, MED SCH, 20090-LUBLIN, 8 JACZEWSKI STR, POLAND
SO American Journal of Human Genetics, (1991) Vol. 49, No. 4 SUPPL, pp. 434.
Meeting Info.: PROCEEDINGS OF THE 8TH INTERNATIONAL CONGRESS OF HUMAN
GENETICS, WASHINGTON, D.C., USA, OCTOBER 6-11, 1991. AM J HUM GENET.
CODEN: AJHGAG. ISSN: 0002-9297.
DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 5 Mar 1992
Last Updated on STN: 5 Mar 1992

L3 ANSWER 37 OF 37 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 8
AN 1988:311405 BIOSIS
DN PREV198886028443; BA86:28443
TI TRANSFORMING GROWTH FACTOR BETA AS A POTENT ***PROMOTER*** IN
TWO-STAGE BALB-C 3T3 CELL TRANSFORMATION.
AU HAMEL E [Reprint author]; KATOH F; MUELLER G; BIRCHMEIER W; YAMASAKI H
CS INTERNATIONAL AGENCY RES CANCER, 150 COURS ALBERT THOMAS, 69372 LYON CEDEX
08, FR
SO Cancer Research, (1988) Vol. 48, No. 10, pp. 2832-2836.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 3 Jul 1988
Last Updated on STN: 3 Jul 1988
AB We have tested transforming growth factor .beta. (TGF.beta.) in the two-stage BALB/c 3T3 cell transformation assay for possible tumor-promoting activity, since it has several effects similar to those of tumor-promoting phorbol ester. After initiation of BALB/c 3T3 cells with 3-methylcholanthrene, treatment with TGF.beta. at 1 ng/ml alone or in combination with epidermal growth factor (EGF) for 4 weeks enhanced the number of transformed foci by 5- to 6-fold in comparison with uninitiated cells. Initiation treatment alone induced no or very few transformed foci in several assays. Treatment with phorbol-12,13-didecanoate (PDD) at 100 ng/ml for 4 weeks enhanced the number of transformed foci in initiated BALB/c 3T3 cells by 4- to 5-fold in comparison with uninitiated cells. Thus, TGF.beta. at 1 ng/ml is as potent as PDD at 100 ng/ml for tumor-promoting activity in the two-stage BALB/c 3T3 cell transformation assay. The enhancing effect of TGF.beta. was dose-related inthe dose range tested (0.03-1 ng/ml) and was not reversible. Some of the foci induced by combined MCA-TGF.beta.-EGF treatment were cloned, and eight out
of nine clones tested produced tumors in nude mice. TGF.beta. (1 ng/ml) plus ***EGF*** (***2*** ng/ml) increased the saturation density to a similar extent as PDD (100 ng/ml) but did not affect the growth of BALB/c 3T3 cells. We observed no change in junctional intercellular communication, as measured by the dye transfer method, when cells were treated with TGF.beta. during the two-stage BALB/c 3T3 cell transformation assay. Nevertheless, there was selective communication between transformed and surrounding nontransformed cells; MCA-TGF.beta. transformed cells intercommunicated among themselves but not with surrounding nontransformed cells. Our results indicate that TGF.beta. has potent tumor-promoting activity in vitro, but that this activity is not mediated by a complete blockage of intercellular communication, as is suggested for phorbol ester tumor promoters.

=> d his

(FILE 'HOME' ENTERED AT 16:15:29 ON 19 JAN 2005)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:15:40 ON 19 JAN 2005
L1 1526 S EGF 2 OR EPITHELIAL GLYCOPROTEIN 2 OR EP CAM OR 17 1A OR GA73
L2 52 S L1 AND (PROMOTER OR REGULA? ELEMENT OR REGULAT? REGION OR 5 U
L3 37 DUP REM L2 (15 DUPLICATES REMOVED)

=> s carcinoma (3a) (select? or restrict? or specific?)
1 FILES SEARCHED...
L4 5817 CARCINOMA (3A) (SELECT? OR RESTRICT? OR SPECIFIC?)

=> s l4 and (promoter or regula? element or regulat? region or 5 UTR)
L5 235 L4 AND (PROMOTER OR REGULA? ELEMENT OR REGULAT? REGION OR 5
UTR)

=> s l5 and lung carcinoma
L6 6 L5 AND LUNG CARCINOMA

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 4 DUP REM L6 (2 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
DUPLICATE 1
AN 2001:225975 BIOSIS
DN PREV200100225975
TI Adenovirus-mediated suicide gene transfer to small cell ***lung*** ***carcinoma*** using a tumor- ***specific*** ***promoter*** .
AU Morimoto, Emiko; Inase, Naohiko [Reprint author]; Miyake, Shuji; Yoshizawa, Yasuyuki
CS Pulmonary Medicine, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo, 113-8519, Japan
ninase.pulm@tmd.ac.jp
SO Anticancer Research, (January-February, 2001) Vol. 21, No. 1A, pp. 329-331. print.
CODEN: ANTRD4. ISSN: 0250-7005.
DT Article
LA English
ED Entered STN: 9 May 2001
Last Updated on STN: 18 Feb 2002
AB The gastrin-releasing peptide (GRP) is expressed in most types of small cell ***lung*** ***carcinoma*** (SCLC) and the GRP ***promoter*** is thought to be potentially useful for tumor-specific expression of the suicide gene in SCLC. We constructed an adenovirus

containing the herpes simplex thymidine kinase suicide gene driven by the GRP ***promoter*** (AdGRP-TK) and transfected it into GRP-expressing SCLC cells (SBC5) to confer sensitivity to ganciclovir (GCV). After infection with AdGRP-TK, SBC5 cells became more sensitive to GCV in vitro. In nude mice, a subcutaneously-inoculated tumor of SBC5 cells infected with AdGRP-TK in advance regressed completely after intraperitoneal administration of GCV. These results suggest that adenovirus-mediated gene transfer of the suicide gene followed by pro-drug treatment may be applicable to SCLC.

L7 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:294518 CAPLUS
DN 135:220767
TI Neuron specific enolase ***promoter*** for suicide gene therapy in small cell ***lung*** ***carcinoma***
AU Tanaka, Michiko; Inase, Naohiko; Miyake, Shuji; Yoshizawa, Yasuyuki
CS Pulmonary Medicine, Tokyo Medical and Dental University, Tokyo, 113-8519, Japan
SO Anticancer Research (2001), 21(1A), 291-294
CODEN: ANTRD4; ISSN: 0250-7005
PB International Institute of Anticancer Research
DT Journal
LA English
AB To investigate the specific transduction of a suicide gene into human small cell ***lung*** ***carcinoma*** (SCLC) cells, we explored the ***promoter*** region of the neuron specific enolase (NSE) gene as a tumor-specific ***promoter*** . In Northern blot anal., NSE mRNA was expressed more abundantly in the SBC3 human SCLC cell line than in the RERF human SCLC cell line, the A549 human lung adenocarcinoma cell line and the HeLa human uterine cervix epitheloid carcinoma cell line. A reporting vector contg. the NSE ***promoter*** (pNSE-LUC) exhibited higher luciferase activity in SBC3 than in the other three cell lines. After transfecting an expression vector contg. the NSE ***promoter*** -bound HSV-TK gene. (pNSE-TK) into the cells, we measured their sensitivity to ganciclovir (GCV). In SBC3, pNSE-TK transfected cells showed about the same sensitivity to GCV as non-transfected (parental) cells. Though the NSE ***promoter*** itself is not optimal for use in suicide gene transfer to SCLC cells, it might be applied as a tumor-specific ***promoter*** after enhancement of its activity.
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:119163 CAPLUS
DN 131:3509
TI Specific point-mutate p53 mini-gene transfecting effects on biological behaviors of a human cancer cell line PG derived from human pulmonary giant carcinoma
AU Xie, Jianwu; Fang, Weigang; Hui, Pei; Li, Baolin; Li, Hongmei; Zhong, Gaogao; Zheng, Jie; Chen, Bifen; Wu, Bingquan
CS Department of Molecular and Biology, Fuzhou Medical University, Fuzhou, 350005, Peop. Rep. China
SO Zhonghua Yixue Zazhi (1999), 79(1), 57-60
CODEN: CHHTAT; ISSN: 0376-2491
PB Zhonghua Yixue Zazhi
DT Journal
LA Chinese
AB The suppressive effects of a murine genomic p53 minigene contg. an Arg-Leu substitution at its encoding amino acid 172 on biol. behaviors of human carcinoma cell were explored and its potential application in cancer gene therapy was evaluated. This mutant p53 gene which lacked of exon 1 and intron 1 expression vector driven by CMV ***promoter*** was co-transfected with PCMVneo into PG cell in which dominant neg. p53 pre-exists by LipofectaMINE and electroporation methods. A wild-type and another kind of genomic mutate-type p53 gene expression vector were transfected. The latter p53 gene encoding protein contained an Arg-His substitution at the same position, and pBLuscript plasmid was used as control. All transfectants were screened by 500 .mu.g/mL geneticin and identified by mouse specific p53 mRNA RT-PCR and Northern blot anal. The biol. behavior changes were studied by colony formation and TUNEL test together with in-situ clone regression for chemosensitivity of anti-cancer drugs after transfection. The transfecting effects of this unusual p53 gene were surprisingly strong. They were more significant than those of the wild-type p53 and could suppress the formation of transgenic colonies and passage. The transgenic colonies were sensitive to be treated in adriamycin and 5-Fu, and the gene transient expression could give cell apoptosis. Codon 172 mutant (Arg-Leu) p53 genomic DNA exhibited a strong suppressive transfecting effects on carcinoma cell, so it was a possible candidate to be used in cancer gene therapy.

L7 ANSWER 4 OF 4 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 92191536 EMBASE
DN 1992191536
TI Identification of a negative ***regulatory*** ***element*** that inhibits c-mos transcription in somatic cells.
AU Zinkel S.S.; Pal S.K.; Szeberenyi J.; Cooper G.M.
CS Dana-Farber Cancer Institute, Harvard Medical School,Boston, MA 02115, United States
SO Molecular and Cellular Biology, (1992) 12/5 (2029-2036).
ISSN: 0270-7306 CODEN: MCEBD4
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English
AB We have used transient expression assays to identify a cis-acting region in the 5' flanking sequence of murine c-mos which, when deleted, allows expression from the c-mos ***promoter*** in NIH 3T3 cells. This negative regulatory sequence, located 400 to 500 nucleotides upstream of the c-mos ATG, also inhibited expression from a heterologous ***promoter*** . In addition to NIH 3T3 cells, the c-mos negative regulatory sequence was active in BALB/3T3 cells, PC12 rat pheochromocytoma cells, and A549 human ***lung*** ***carcinoma*** cells. Site- ***specific*** mutagenesis identified three possibly interacting regions that were involved in negative regulatory activity, located around -460, -425, and -405 with respect to the ATG. RNase protection analysis indicated that once the negative regulatory sequences were deleted, transcription in NIH 3T3 cells initiated from the same transcription initiation sites normally utilized in spermatocytes, approximately 280 nucleotides upstream of the ATG. Deletions beyond the spermatocyte ***promoter*** , however, allowed transcription initiation from progressively downstream c-mos sequences. Deletion or mutation of sequences surrounding the oocyte ***promoter*** at -53 also had little effect on expression of c-mos constructs in NIH 3T3 cells. Therefore, the major determinant of c-mos expression in NIH 3T3 cells was removal of the negative regulatory sequence rather than the utilization of a unique ***promoter*** . The c- mos negative regulatory sequences thus appear to play a significant role in tissue-specific c-mos expression by inhibiting transcription in somatic cells.

=> d his

(FILE 'HOME' ENTERED AT 16:15:29 ON 19 JAN 2005)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:15:40 ON 19 JAN 2005
L1 1526 S EGF 2 OR EPITHELIAL GLYCOPROTEIN 2 OR EP CAM OR 17 1A OR GA73
L2 52 S L1 AND (PROMOTER OR REGULA? ELEMENT OR REGULAT? REGION OR 5 U
L3 37 DUP REM L2 (15 DUPLICATES REMOVED)
L4 5817 S CARCINOMA (3A) (SELECT? OR RESTRICT? OR SPECIFIC?)
L5 235 S L4 AND (PROMOTER OR REGULA? ELEMENT OR REGULAT? REGION OR 5
L6 6 S L5 AND LUNG CARCINOMA
L7 4 DUP REM L6 (2 DUPLICATES REMOVED)

=> s l5 and lung
L8 24 L5 AND LUNG

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9 20 DUP REM L8 (4 DUPLICATES REMOVED)

=> s l9 not l7
L10 16 L9 NOT L7

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 16 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 16 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 2003:106886 BIOSIS
DN PREV200300106886
TI The variant hepatocyte nuclear factor 1 activates the P1 ***promoter*** of the human alpha-folate receptor gene in ovarian carcinoma.
AU Tomassetti, Antonella [Reprint Author]; Mangiarotti, Fabio; Mazzi, Mimma; Sforzini, Sabrina; Miotti, Silvia; Galmozzi, Enrico; Elwood, Patrick C.; Canevari, Silvana
CS Unit of Molecular Therapies, Department of Experimental Oncology, Istituto Nazionale Tumori, Via Venezian 1, 20133, Milan, Italy antonella.tomassetti@istitutotumori.mi.it
SO Cancer Research, (February 1 2003) Vol. 63, No. 3, pp. 696-704. print.
ISSN: 0008-5472 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 26 Feb 2003
Last Updated on STN: 26 Feb 2003
AB The alpha folate receptor (alphaFR) is a membrane glycoprotein that binds folates, and mediates their uptake and that of antifolate drugs. alphaFR is absent on ovarian surface epithelium (OSE) but is detectable during early transforming events in this epithelium, with increasing expression levels in association with tumor progression. Analysis of transcriptional regulation of the alphaFR gene have revealed two ***promoter*** regions, P1 and P4, flanking exons 1 and 4, respectively, and a requirement for three SP1 sites and an INR element for optimal P4 activity. Here, we focused on the P1 transcription regulation in ovarian carcinoma cells. RNase protection assay indicated that the 5'-untranslated region is heterogeneous because of different start sites and alternative splicing of exon 3. A core region of the P1 ***promoter*** was sufficient for maximal ***promoter*** activity in

ovarian carcinoma cell lines but not in OSE cells or in alphaFR-nonexpressing cell lines. Deletion and mutation analysis of this core ***promoter*** identified a cis- ***regulatory*** ***element*** at position +27 to +33 of the untranslated exon 1, which is responsible for maximum P1 activity. This element formed an abundant DNA-protein complex with nuclear proteins from ovarian cancer cells but not from other cell lines or OSE cells. Competition experiments and supershift assays demonstrated binding of the P1 cis- ***regulatory*** ***element*** by a transcription factor involved in embryonic development, the variant hepatocyte nuclear factor-1 (vHNF1). Analysis of RNA from various cell lines and surgical specimens confirmed that vHNF1 is expressed in ovarian carcinomas. Thus, vHNF1 regulates tissue- ***specific*** transcription in ovarian ***carcinoma*** .

L10 ANSWER 2 OF 16 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 1999:484497 BIOSIS
DN PREV199900484497
TI DNA vaccination against the ovarian carcinoma-associated antigen folate receptor alpha (FRalpha) induces cytotoxic T lymphocyte and antibody responses in mice.
AU Neglia, Francesca; Orengo, Anna Maria; Cilli, Michele; Meazza, Raffaella; Tomassetti, Antonella; Canevari, Silvana; Melani, Cecilia; Colombo, Mario P.; Ferrini, Silvano [Reprint author]
CS Centro di Biotecnologie Avanzate, Istituto Nazionale per la Ricerca sul Cancro, Largo Rosanna Benzi No. 10, 16132, Genova, Italy
SO Cancer Gene Therapy, (July-Aug., 1999) Vol. 6, No. 4, pp. 349-357. print.
ISSN: 0929-1903.
DT Article
LA English
ED Entered STN: 16 Nov 1999
Last Updated on STN: 16 Nov 1999
AB Human folate receptor alpha (FRalpha) is a folate-binding protein that is ***selectively*** overexpressed in ovarian ***carcinoma*** and has been regarded as a suitable target antigen for immunotherapy purposes. To study the possible use of this antigen in DNA vaccination, FRalpha cDNA was ligated into the VR1012 (Vical) expression vector under the transcriptional control of the cytomegalovirus ***promoter*** . A total of 100 mug of purified plasmid DNA was injected intramuscularly in BALB/c mice three times at 14-day intervals. At 10 days after the second injection, the sera of the animals (100%) displayed significant antibody titers (by indirect immunofluorescence and fluorescence-activated cell sorter analysis) against syngeneic C26 cells transduced with FRalpha, but not against unmodified C26 cells. Immunoglobulin G2a was the predominant isotype. In addition, specific cytotoxic T lymphocyte activity against FRalpha-transduced C26 cells could be detected in splenocytes from all immunized animals. Coinjection of a plasmid containing interleukin-2 cDNA increased both antibody titers and cytotoxic T lymphocyte activity. Challenge by subcutaneous injection with FRalpha-transduced C26 cells (performed 10 days after the third injection) showed a statistically significant delay in tumor growth. Vaccination with the FRalpha and interleukin-2 cDNA mixture, which was performed after an intravenous injection of FRalpha-transduced cells, enhanced the mean survival time and reduced the number of ***lung*** metastases, thus suggesting that such vaccination is effective even against preexisting tumor cells.

L10 ANSWER 3 OF 16 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2001365405 EMBASE
TI Molecular detection of p16 ***promoter*** methylation in the serum of patients with esophageal squamous cell carcinoma.
AU Hibi K.; Taguchi M.; Nakayama H.; Takase T.; Kasai Y.; Ito K.; Akiyama S.; Nakao A.
CS K. Hibi, Second Department of Surgery, Nagoya University School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466-8550, Japan. khibi@med.nagoya-u.ac.jp
SO Clinical Cancer Research, (2001) 7/10 (3135-3138).
Refs: 19
ISSN: 1078-0432 CODEN: CCREF4
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
009 Surgery
016 Cancer
022 Human Genetics
048 Gastroenterology
LA English
SL English
AB Purpose and Experimental Design: Recent evidence shows that the presence of ***promoter*** hypermethylation of tumor suppressor genes has been demonstrated in the serum DNA of patients with various cancers such as ***lung*** , liver, and head and neck cancer. We have examined ***promoter*** hypermethylation of the p16 gene in esophageal squamous cell ***carcinoma*** (SCC) using methylation- ***specific*** PCR to detect tumor DNA in the serum. Results: Aberrant ***promoter*** methylation of the p16 gene was detected in 31 of 38 (82%) esophageal SCCs. Subsequently, we tested for ***promoter*** methylation in the paired serum DNA of 31 patients with a p16 alteration in the primary tumor. We found that 7 of these 31 (23%) patients had the same methylation changes in the serum DNA. Conclusions: This result indicates that ***promoter*** methylation present in the tumors of esophageal SCC patients can be detected in the serum of the same patient and that this approach can potentially be used for the screening and monitoring of the disease.

L10 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:930659 CAPLUS
TI Cancer-specific activation of the survivin ***promoter*** and its potential use in gene therapy
AU Chen, Jin-Shing; Liu, Jaw-Ching; Shen, Lei; Rau, Kung-Ming; Kuo, Hsu-Ping; Li, Yan M.; Shi, Daren; Lee, Yung-Chie; Chang, King-Jen; Hung, Mien-Chie.
CS Department of Molecular and Cellular Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX, 77030, USA
SO Cancer Gene Therapy (2004), 11(11), 740-747
CODEN: CGTHEG; ISSN: 0929-1903
PB Nature Publishing Group
DT Journal
LA English
AB Survivin is expressed in many cancers but not in normal adult tissues and is transcriptionally regulated. To test the feasibility of using the survivin ***promoter*** to induce cancer-specific transgene expression in ***lung*** cancer gene therapy, a vector expressing a luciferase gene driven by the survivin ***promoter*** was constructed and evaluated in vitro and in vivo. It was found that the survivin ***promoter*** was generally more highly activated in cancer cell lines than in normal and immortalized normal cell lines. When delivered i.v. by DNA:liposome complexes, the survivin ***promoter*** was more than 200 times more cancer specific than the cytomegalovirus ***promoter*** in vivo. To identify ***lung*** cancer patients who may benefit from gene therapy with the survivin ***promoter*** , survivin protein expression was measured in surgical specimens of 75 non-small-cell ***lung*** cancers and 10 normal ***lung*** tissues by immunohistochem. staining and found that survivin is expressed in most of the non-small-cell ***lung*** cancers tested (81%, 61 of 75) but none of the normal ***lung*** tissues. The survivin ***promoter*** also induced transgene expression of a mutant Bik in cancer cells, which suppressed the growth of cancer cells in vitro and in vivo. These results indicate that the survivin ***promoter*** is a cancer-specific ***promoter*** for various cancers and that it may be useful in cancer gene therapy.
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:413860 CAPLUS
DN 139:917
TI Dual specificity tumor killing viral vectors driven by the telomerase ***promoter*** and uses for cancer gene therapy
IN Irving, John M.; Karpf, David B.; Schiff, J. Michael
PA USA
SO U.S. Pat. Appl. Publ., 25 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003099616	A1	20030529	US 2002-206447	20020725
PRAI US 2001-308029P	P	20010725		

AB The present invention discloses the specificity of multiple transcriptional regulatory elements can be combined to make adenoviral vector systems that selectively target cancer cells and its uses in gene therapy for cancers. The ***promoter*** for telomerase reverse transcriptase (TERT) can be combined in a remarkably synergistic fashion with another ***promoter*** that has expression restricted to cancer cells or a particular tissue type. The two promoters work synergistically for exquisite targeting of the malignant cells-where it causes cell lysis while leaving neighboring healthy cells intact. This invention also includes methods for constructing and selecting the viral vectors, host cells transduced with the vector construct, and the host cells monitored for any effect of the vector.

L10 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:178335 CAPLUS
DN 138:231393
TI Tumor-specific gene therapy for undifferentiated thyroid carcinoma using the human telomerase reverse transcriptase ***promoter***
AU Takeda, Teiji; Hashizume, Kiyoshi
CS Department of Aging Medicine and Geriatrics, Shinshu University School of Medicine, Japan
SO Horumon to Rinsho (2003), 51(2), 149-154
CODEN: HORIAE; ISSN: 0045-7167
PB Igaku no Sekaisha
DT Journal
LA Japanese
AB The authors previously developed recombinant adenoviruses carrying herpes simplex virus thymidine kinase (HSVtk) genes to evaluate the possibility of tissue-specific gene therapy for thyroid carcinoma. The HSVtk gene was driven by a minimal thyroglobulin (TG) ***promoter*** (AdTGtk) and a tandemly repeated minimal TG ***promoter*** (Ad2.times.TGtk) to obtain thyroid-specific cell killing ability. Ad2.times.TGtk showed a beneficial effect for tissue-specific gene therapy for TG-producing thyroid carcinoma, but not for undifferentiated thyroid carcinoma. The authors

placed HSVtk gene under the control of human telomerase reverse transcriptase (hTERT) gene ***promoter*** (AdhTERTtk). Tumor-specific transcriptional activity by hTERT ***promoter*** was confirmed. The transduction of HSVtk genes by infection with AdhTERTtk followed by ganciclovir (GCV) treatment showed powerful cytotoxicity for TG-producing and non-TG-producing thyroid carcinoma cell lines but no or little cytotoxicity for normal cell lines. After adenovirus/GCV treatment for ARO tumor-bearing nude mice, AdhTERTtk inhibited the tumor growth. Ad2.times.TGtk/GCV and AdhTERTtk/GCV treatment showed no or very little cytotoxicity in liver, kidney, spleen, thyroid, ***lung*** , and testis. These data suggest a beneficial effect of AdhTERTtk for gene therapy of undifferentiated thyroid carcinoma without toxicity for normal tissues.

L10 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:897718 CAPLUS
DN 138:120721
TI Characterization of a tissue-specific CDP/Cux isoform, p75, activated in breast tumor cells
AU Goulet, Brigitte; Watson, Peter; Poirier, Madeleine; Leduy, Lam; Berube, Ginette; Meterissian, Sarkis; Jolicoeur, Paul; Nepveu, Alain
CS Molecular Oncology Group, McGill University Health Center, Montreal, QC, H3A 1A1, Can.
SO Cancer Research (2002), 62(22), 6625-6633
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB Two isoforms of the CCAAT-displacement protein/cut homeobox (CDP/Cux) transcription factor have been characterized thus far. The full length protein, p200, which contains four DNA binding domains, transiently binds to DNA and carries the CCAAT-displacement activity. The p110 isoform is generated by proteolytic processing at the G1-S transition and is capable of stable interaction with DNA. Here the authors demonstrate the existence of a shorter CDP/Cux isoform, p75, which contains only two DNA binding domains, Cut repeat 3 and the Cut homeodomain, and binds more stably to DNA. CDP/Cux p75 was able to repress a reporter carrying the ***promoter*** for the cyclin-dependent kinase inhibitor p21 gene and to activate a DNA polymerase .alpha. gene reporter. Expression of CDP/Cux p75 involved a novel mechanism: transcription initiation within intron 20. The intron 20-initiated mRNA (I20-mRNA) was expressed at higher level in the thymus and in CD4+/CD8+ and CD4+ T cells. I20-mRNA was expressed only
weakly or not at all in normal human mammary epithelial cells and normal breast tissues but was detected in many breast tumor cells lines and breast tumors. In invasive tumors a significant assocn. was established between higher I20-mRNA expression and a diffuse infiltrative growth pattern. In agreement with these findings, T47D breast cancer cells stably expressing p75 could not form tubule structures in collagen but rather developed as solid undifferentiated aggregates of cells. Taken together, these results suggest that aberrant expression of the CDP/Cux p75 isoform in mammary epithelial cells may be assocd. with the process of tumorigenesis in breast cancer.
RE.CNT 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:393088 CAPLUS
DN 137:107256
TI Tumour specific ***promoter*** region methylation of the human homologue of the Drosophila Roundabout gene DUTT1 (ROBO1) in human cancers
AU Dallol, Ashraf; Forgacs, Eva; Martinez, Alonso; Sekido, Yoshitaka; Walker, Rosemary; Kishida, Takeshi; Rabbitts, Pamela; Maher, Eamonn R.; Minna, John D.; Latif, Farida
CS Section of Medical and Molecular Genetics, Department of Paediatrics and Child Health, The Medical School, University of Birmingham, Birmingham, B15 2TT, UK
SO Oncogene (2002), 21(19), 3020-3028
CODEN: ONCNES; ISSN: 0950-9232
PB Nature Publishing Group
DT Journal
LA English
AB The human homolog of the Drosophila Roundabout gene DUTT1 (Deleted in U
Twenty Twenty) or ROBO1 (Locus Link ID 6091), a member of the NCAM family of receptors, was recently cloned from the ***lung*** cancer tumor suppressor gene region 2 (LCTSGR2 or U2020 region) at 3p12. DUTT1 maps within a region of overlapping homozygous deletions characterized in both small cell ***lung*** cancer lines (SCLC) and in a breast cancer line. In this report the authors (a) defined the genomic organization of the DUTT1 gene, (b) performed mutation and expression anal. of DUTT1 in ***lung*** , breast and kidney cancers, (c) identified tumor specific ***promoter*** region methylation of DUTT1 in human cancers. The gene was found to contain 29 exons and spans at least 240 kb of genomic sequence. The 5' region contains a CpG island, and the poly(A)+ tail has an atypical 5'-GATAAA-3' signal. The authors analyzed DUTT1 for mutations in ***lung*** , breast and kidney cancers; no inactivating mutations were detected by PCR-SSCP. However, seven germline missense changes were
found and characterized. DUTT1 expression was not detectable in one out of 18 breast tumor lines analyzed by RT-PCR. Bisulfite sequencing of the ***promoter*** region of DUTT1 gene in the HTB-19 breast tumor cell line (not expressing DUTT1) showed complete hypermethylation of CpG sites within the ***promoter*** region of the DUTT1 gene (-244 to +27 relative to the translation start site). The expression of DUTT1 gene was reactivated in HTB-19 after treatment with the demethylating agent 5-aza-2'-deoxycytidine. The same region was also hypermethylated in six out of 32 (19%) primary invasive breast carcinomas and eight out of 44 (18%) primary clear cell renal cell carcinomas (CC-RCC) and in one out of 26 (4%) primary NSCLC tumors. Furthermore 80% of breast and 75% of CC-RCC
tumors showing DUTT1 methylation had allelic losses for 3p12 markers hence obeying Knudson's two hit hypothesis. The authors' findings suggest that DUTT1 warrants further anal. as a candidate for the tumor suppressor gene (TSG) at 3p12, a region defined by hemi and homozygous deletions and functional anal.
RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:654737 CAPLUS
DN 135:206499
TI Non-squamous epithelium-specific EGP-2 ***promoter*** driven transcription for cancer therapy
IN De Leij, Lou Franciscus Maria Hubertus; McLaughlin, Pamela Marijke Jane; Ruiters, Marcel Herman Josef; Harmsen, Martin Conrad; Van der Molen, Henk; Terpstra, Peter; Dokter, Willem Hendrik Abraham
PA Rijksuniversiteit te Groningen, Neth.
SO Eur. Pat. Appl., 21 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1130106	A1	20010905	EP 2000-200728	20000301
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
CA 2364314	AA	20010927	CA 2001-2364314	20010228
WO 2001071015	A2	20010927	WO 2001-NL166	20010228
WO 2001071015	A3	20020131		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1190085	A2	20020327	EP 2001-952047	20010228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
NZ 515206	A	20040326	NZ 2001-515206	20010228
US 2002156041	A1	20021024	US 2002-9579	20020326
PRAI EP 2000-200728	A	20000301		
WO 2001-NL166	W	20010228		

AB The invention relates to the field of cancer therapy and diagnosis, in particular of carcinomas. The invention provides an isolated and/or recombinant nucleic acid comprising a tissue specific ***promoter*** or functional fragment thereof allowing for expression of a nucleic acid of interest operably linked to said ***promoter*** or functional fragment thereof in a cancer cell wherein said expression in said cancer cell is essentially ***carcinoma*** ***selective*** . In a preferred embodiment, the invention provides the isolation and use of EGP-2 transcriptional regulatory sequences to regulate transient expression of the cytosine deaminase gene in EGP-2 expressing carcinoma cells. The invention further provides a vector or gene delivery vehicle comprising a nucleic acid according to the invention. Such gene delivery vehicles as provided by the invention are very useful in carcinoma therapy, or in therapy directed at non-squamous epithelium.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:380649 CAPLUS
DN 135:4472
TI Antigen-binding fragments specific for dendritic cells, compositions and methods of use thereof antigens recognized thereby and cells obtained thereby
IN Schmitz, Juergen; Dzionek, Andrzej; Buck, David William
PA Miltenyi Biotech G.m.b.H., Germany
SO PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001036487	A2	20010525	WO 2000-IB1832	20001115
WO 2001036487	A3	20020510		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,				

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
CA 2396428 AA 20010525 CA 2000-2396428 20001115
EP 1301539 A2 20030416 EP 2000-979855 20001115
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR
JP 2004512006 T2 20040422 JP 2001-538976 20001115
PRAI US 1999-165555P P 19991115
US 1999-167076P P 19991123
US 2000-179003P P 20000128
US 2000-180775P P 20000207
US 2000-196824P P 20000411
US 2000-197205P P 20000413
WO 2000-IB1832 W 20001115
AB The invention provides antigen-binding fragments specific for dendritic cells and effective in treatment and/or diagnosing a variety of disorders. Methods of use are also provided as are methods for screening for addnl. such antigen-binding fragments and the products obtained thereby.

L10 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:310088 CAPLUS
DN 134:362609
TI p16INK4a and histology-specific methylation of CpG islands by exposure to tobacco smoke in non-small cell ***lung*** cancer
AU Kim, Duk-Hwan; Nelson, Heather H.; Wiencke, John K.; Zheng, Shichun; Christiani, David C.; Wain, John C.; Mark, Eugene J.; Kelsey, Karl T.
CS Department of Environmental Health, Harvard School of Public Health, Boston, MA, 02115, USA
SO Cancer Research (2001), 61(8), 3419-3424
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB The p16INK4a protein inhibits cyclin-dependent kinase 4, a key regulator of progression through the G1 phase of the cell cycle. Methylation of CpG islands in the ***promoter*** region is an important avenue for inactivation of p16. The mechanism of methylation of the p16 ***promoter*** region, however, has not been elucidated. Recent reports investigating p16 methylation in non-small cell ***lung*** cancer (NSCLC) suggest that carcinogens in tobacco smoke induce the DNA methylation process. We investigated the assocn. between methylation of the p16 ***promoter*** region and exposure to tobacco smoke in 185 primary NSCLCs. We also studied the relationship of p16 methylation with mutation of the K-ras and p53 genes, as well as with methylation at the DAP-kinase and p14ARF loci. Finally, we evaluated the prognostic significance of p16 methylation in NSCLC. The prevalence of p16 methylation was greater in squamous cell carcinoma (41%) compared with adenocarcinoma (22%; P = 0.03; Fisher's exact test). Methylation of p16 was significantly assocd. with pack-years smoked (P = 0.007; Wilcoxon rank sum test), duration of smoking (P = 0.0009; Wilcoxon rank sum test), and neg. with the time since quitting smoking (P = 0.03; Wilcoxon rank sum test). No methylation of the nearby p14ARF locus was detected, and methylation of the DAP-kinase locus was not assocd. with either p16 methylation or with exposure to tobacco smoke. In patients with stage 1 adenocarcinoma, p16 methylation was an independent risk factor predicting significantly shorter postsurgery survival (P = 0.03), controlling for the significant effects of other factors, including K-ras mutation. These findings suggest that methylation of CpG islands in tobacco-assocd. cancers occurs in a gene- and tissue-specific manner and is induced directly or indirectly by exposure to tobacco smoke in NSCLC.
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:247215 CAPLUS
DN 134:276498
TI Engineering of replication selective adenoviruses with tumor-associated antigen ***promoter*** for use in cancer therapy
IN Molnar-kimber, Katherine; Toyoizumi, Takane
PA The Trustees of the University of Pennsylvania, USA
SO PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE
PI WO 2001023004 A1 20010405 WO 2000-US27212 20001002
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRAI US 1999-157224P P 19990930
AB The invention provides a replication selective adenovirus (Ad) mutant with improved selectively for tumor cells expressing the tumor assocd. antigen in cancers and malignancies, as well as in proliferative cells, characterizing diseases, such as restenosis,intimal proliferative disease and pulmonary hypertension. The selected Ad vectors are driven by promoters of the tumor assocd. antigens, or RNA transcripts or genes therefor, substituting for the activity of at least adenovirus E1A ***promoter*** , which has been deactivated or diminished. Also provided is the use of the Ad vector to deliver therapeutic compns. to patients, as well as a method for treating cancers, such as CEA pos. cancers, or proliferative cell diseases in a patient by administering to the patient an effective amt. of the Ad vector, which may also express a therapeutic gene or peptide, and treatment may also be combined with radiation, chemotherapy or immunomodulatory agents. The Ad is designed to replicate within the tumor cell, thereby spreading throughout the tumor nodule. This permits the delivery of a much higher dose of the heterologous therapeutic protein than previously possible, and the virus achieves a direct, oncolytic effect on the tumor.
RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:582108 CAPLUS
DN 132:120711
TI Tumour-specific arginine vasopressin ***promoter*** activation in small-cell ***lung*** cancer
AU Coulson, J. M.; Stanley, J.; Woll, P. J.
CS CRC Department of Clinical Oncology, University of Nottingham, Nottingham, NG5 1PB, UK
SO British Journal of Cancer (1999), 80(12), 1935-1944
CODEN: BJCAAI; ISSN: 0007-0920
PB Churchill Livingstone
DT Journal
LA English
AB Small-cell ***lung*** cancer (SCLC) can produce numerous mitogenic neuropeptides, which are not found in normal respiratory epithelium. Arginine vasopressin is detected in up to two-thirds of SCLC tumors whereas normal physiol. expression is essentially restricted to the hypothalamus. This presents the opportunity to identify elements of the gene ***promoter*** which could be exploited for SCLC-specific targeting. A series of human vasopressin 5' ***promoter*** fragments (1048 bp, 468 bp and 199 bp) were isolated and cloned upstream of a reporter gene. These were transfected into a panel of ten cell lines, including SCLC with high or low endogenous vasopressin transcription, non-SCLC and bronchial epithelium. All these fragments directed reporter gene expression in the five SCLC cell lines, but had negligible activity in the control lines. The level of reporter gene expression reflected the level of endogenous vasopressin prodn., with up to 4.9-fold (s.d. 0.34) higher activity than an SV40 ***promoter*** . The elements required for this strong, restricted, SCLC-specific ***promoter*** activity are contained within the 199-bp fragment. Further anal. of this region indicated involvement of E-box transcription factor binding sites, although tumor-specificity was retained by a 65-bp minimal ***promoter*** fragment. These data show that a short region of the vasopressin ***promoter*** will drive strong expression in SCLC in vitro and raise the possibility of targeting gene therapy to these tumors.
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:244743 CAPLUS
DN 130:276738
TI Inducing tumor-specific cytotoxicity using vectors containing H19 or insulin-like growth factor gene regulatory elements
IN Hochberg, Abraham; Ayesh, Suhail
PA Yissum Research Development Company of the Hebrew University of Jerusalem, Israel
SO PCT Int. Appl., 67 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4
PATENT NO. KIND DATE APPLICATION NO. DATE
PI WO 9918195 A2 19990415 WO 1998-IL486 19981004
WO 9918195 A3 19990812
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
CA 2308124 AA 19990415 CA 1998-2308124 19981004
AU 9894571 A1 19990427 AU 1998-94571 19981004
AU 755774 B2 20021219
EP 1019499 A2 20000719 EP 1998-947759 19981004

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
BR 9812717 A 20000822 BR 1998-12717 19981004
JP 2001519148 T2 20011023 JP 2000-514993 19981004
RU 2214280 C2 20031020 RU 2000-111553 19981004
NO 2000001684 A 20000602 NO 2000-1684 20000331
PRAI US 1997-943608 A 19971003
WO 1998-IL486 W 19981004
AB The invention relates to the specific expression of heterologous sequences, particularly genes encoding cytotoxic products, in tumor cells under the control of regulatory transcriptional sequences. Particularly preferred promoters include H19 regulatory sequences, the IGF-1 ***promoter*** , and the IGF-2 P3 and P4 promoters from genomically imprinted genes. The invention provides expression constructs and methods of administering such expression constructs. The H19 regulatory sequences facilitate expression of a heterologous gene in five different bladder cancer cell lines (HT-1376, EJ28, T24P, 1197, and UM-UC-3). When transfected into bladder cancer cell, an H19/HSV-TK expression plasmid induces bladder cancer cell-specific cytotoxicity in the presence of ganciclovir. The compns. and methods of the invention are useful in the treatment of cancer.

L10 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:793064 CAPLUS
DN 130:35133
TI P-selectin translocation to vascular epithelial lumen by ionizing radiation, and therapeutic use
IN Hallahan, Dennis E.; Virudachalam, Subbulakshmi
PA Arch Development Corporation, USA
SO PCT Int. Appl., 178 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9853852	A1	19981203	WO 1998-US10913	19980529

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA 2290563	AA	19981203	CA 1998-2290563	19980529
AU 9886570	A1	19981230	AU 1998-86570	19980529
EP 986401	A1	20000322	EP 1998-937941	19980529
EP 986401	B1	20040225		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

AT 260116	E	20040315	AT 1998-937941	19980529

PRAI US 1997-48141P P 19970530
WO 1998-US10913 W 19980529
AB The present invention relates to the use of P-selectin as a targeting agent in radiotherapies for vascular related disease. P-selectin is translocated to the lumen of vascular endothelia as a result of radiation. Thus, P-selecting provides a target for receptor-mediated delivery of drugs, including anticancer drugs and drugs for the treatment of vascular disease. However, P-selectin also plays a role in the activation of certain inflammatory cells and, as such, plays a role in radiation-induced inflammation. By interfering with P-selectin induction of inflammation, it is possible to modulate inflammatory responses to radiation therapy.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:210872 CAPLUS
DN 128:266956
TI Hex II tumor-specific ***promoter*** and its use in gene-targeted cancer therapy
IN Batist, Gerald; Katabi, Maha
PA McGill University, Can.; Batist, Gerald; Katabi, Maha
SO PCT Int. Appl., 20 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9813507	A1	19980402	WO 1997-CA691	19970922

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA 2266846	AA	19980402	CA 1997-2266846	19970922
AU 9742927	A1	19980417	AU 1997-42927	19970922
EP 954590	A1	19991110	EP 1997-918865	19970922

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

US 2001011128	A1	20010802	US 2000-739223	20001219

PRAI US 1996-26678P P 19960925
WO 1997-CA691 W 19970922
US 1999-276005 A2 19990325
AB The present invention relates to a tumor-specific ***promoter*** , the Hex II ***promoter*** , for use in gene targeted therapy that is differentially regulated in cancer cells. The present invention also relates to a gene construct, which includes the Hex II ***promoter*** in a vector selected from pCAT basic expression vector p.DELTA.ElspIB, called pHexII4557-CAT, and a shuttle plasmid which includes either .beta.-gal or HSV Tk, called p.DELTA.ElspIBHex-LacZ and p.DELTA.ElspIBHex-TK.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> LOG Y

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		264.84	265.05

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	ENTRY	SINCE FILE SESSION	TOTAL
CA SUBSCRIBER PRICE		-31.39	-31.39

STN INTERNATIONAL LOGOFF AT 16:57:30 ON 19 JAN 2005

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NEWS 5 NOV 30 PHAR reloaded with additional data
NEWS 6 DEC 01 LISA now available on STN
NEWS 7 DEC 09 12 databases to be removed from STN on December 31, 2004
NEWS 8 DEC 15 MEDLINE update schedule for December 2004
NEWS 9 DEC 17 ELCOM reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS 10 DEC 17 COMPUAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS 11 DEC 17 SOLIDSTATE reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS 12 DEC 17 CERAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS 13 DEC 17 THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
NEWS 14 DEC 30 EPFULL: New patent full text database to be available on STN
NEWS 15 DEC 30 CAPLUS - PATENT COVERAGE EXPANDED
NEWS 16 JAN 03 No connect-hour charges in EPFULL during January and February 2005
NEWS 17 JAN 11 CA/CAPLUS - Expanded patent coverage to include Russia (Federal Institute of Industrial Property)

NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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FILE 'HOME' ENTERED AT 17:01:43 ON 19 JAN 2005

=> FIL BIOSIS EMBASE CAPLUS
COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.21 0.21

FILE 'BIOSIS' ENTERED AT 17:01:49 ON 19 JAN 2005
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=> s lung carcinoma (3a)(select? or specific? or restric?)
L1 248 LUNG CARCINOMA (3A)(SELECT? OR SPECIFIC? OR RESTRIC?)

=> s l1 and (promoter or regulat? region or regulat? element or 5 UTR)
L2 4 L1 AND (PROMOTER OR REGULAT? REGION OR REGULAT? ELEMENT OR 5 UTR)

=> dup rem l2\
ENTER L# LIST OR (END):l2
'L2\' IS NOT VALID. VALID FILE NAMES ARE 'BIOSIS, EMBASE, CAPLUS'
You have entered a file name of duplicates to keep that is not referenced by any of the L#s specified for this DUPLICATE command.
The file names of duplicates that can be kept are listed above.
Please enter one of these file names.

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 2 DUP REM L2 (2 DUPLICATES REMOVED)

=> d bib abs

L3 ANSWER 1 OF 2 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
DUPLICATE 1
AN 2001:225975 BIOSIS
DN PREV200100225975
TI Adenovirus-mediated suicide gene transfer to small cell ***lung*** ***carcinoma*** using a tumor- ***specific*** ***promoter*** .
AU Morimoto, Emiko; Inase, Naohiko [Reprint author]; Miyake, Shuji; Yoshizawa, Yasuyuki
CS Pulmonary Medicine, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo, 113-8519, Japan
ninase.pulm@tmd.ac.jp
SO Anticancer Research, (January-February, 2001) Vol. 21, No. 1A, pp. 329-331. print.
CODEN: ANTRD4. ISSN: 0250-7005.
DT Article
LA English
ED Entered STN: 9 May 2001
Last Updated on STN: 18 Feb 2002
AB The gastrin-releasing peptide (GRP) is expressed in most types of small cell lung carcinoma (SCLC) and the GRP ***promoter*** is thought to be potentially useful for tumor-specific expression of the suicide gene in SCLC. We constructed an adenovirus containing the herpes simplex thymidine kinase suicide gene driven by the GRP ***promoter*** (AdGRP-TK) and transfected it into GRP-expressing SCLC cells (SBC5) to confer sensitivity to ganciclovir (GCV). After infection with AdGRP-TK, SBC5 cells became more sensitive to GCV in vitro. In nude mice, a subcutaneously-inoculated tumor of SBC5 cells infected with AdGRP-TK in advance regressed completely after intraperitoneal administration of GCV. These results suggest that adenovirus-mediated gene transfer of the suicide gene followed by pro-drug treatment may be applicable to SCLC.

=> d bib abs 2

L3 ANSWER 2 OF 2 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 92191536 EMBASE
DN 1992191536
TI Identification of a negative ***regulatory*** ***element*** that inhibits c-mos transcription in somatic cells.
AU Zinkel S.S.; Pal S.K.; Szeberenyi J.; Cooper G.M.
CS Dana-Farber Cancer Institute, Harvard Medical School,Boston, MA 02115, United States
SO Molecular and Cellular Biology, (1992) 12/5 (2029-2036).
ISSN: 0270-7306 CODEN: MCEBD4
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English
AB We have used transient expression assays to identify a cis-acting region in the 5' flanking sequence of murine c-mos which, when deleted, allows expression from the c-mos ***promoter*** in NIH 3T3 cells. This negative regulatory sequence, located 400 to 500 nucleotides upstream of the c-mos ATG, also inhibited expression from a heterologous ***promoter*** . In addition to NIH 3T3 cells, the c-mos negative regulatory sequence was active in BALB/3T3 cells, PC12 rat pheochromocytoma cells, and A549 human ***lung*** ***carcinoma*** cells. Site- ***specific*** mutagenesis identified three possibly interacting regions that were involved in negative regulatory activity, located around -460, -425, and -405 with respect to the ATG. RNase protection analysis indicated that once the negative regulatory sequences were deleted, transcription in NIH 3T3 cells initiated from the same transcription initiation sites normally utilized in spermatocytes, approximately 280 nucleotides upstream of the ATG. Deletions beyond the spermatocyte ***promoter*** , however, allowed transcription initiation from progressively downstream c-mos sequences. Deletion or mutation of sequences surrounding the oocyte ***promoter*** at -53 also had little effect on expression of c-mos constructs in NIH 3T3 cells. Therefore, the major determinant of c-mos expression in NIH 3T3 cells was removal of the negative regulatory sequence rather than the utilization of a unique ***promoter*** . The c- mos negative regulatory sequences thus appear to play a significant role in tissue-specific c-mos expression by inhibiting transcription in somatic cells.

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---Logging off of STN---

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Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 44.62 44.83

STN INTERNATIONAL LOGOFF AT 17:12:01 ON 19 JAN 2005

---Logging off of STN---

END

Unable to generate the STN prompt.
Exiting the script...